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PRESIDENTIAL ADDRESS AT THE SOUTH AFRICAN MEDICAL CONGRESS, DURBAN

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President of the Medical Association of South Africa

Dr. Grant-Whyte expressed regret at the absence owing to indisposition of His Excellency the Governor General, Dr. E. H. Jansen, Patron of Congress, and welcomed in his place His Honour the Administrator of Natal, Dr. D. G. Shepstone, Chancellor of the University of Natal. He went on to express his appreciation of his election as President of Congress and President of the Medical Association of South Africa, and paid a tribute to his wife for her help, forbearance and understanding. He referred with satisfaction to the distinguished company of visitors from overseas who were honouring the Congress with their presence, and proceeded to say:

THE ATOMIC AGE

Most of us here are, in a very real sense, products of the pre-atomic age. We are nineteenth century men. Our university education was taken towards the end of a scientific era which closed with the discovery of the nature of the atom, the amendment to Dalton's atomic theory, the overthrow of the text-book axiom that the atom was indivisible.

This discovery is the greatest single discovery since Isaac Newton. It, and the energies released by this single fact, are changing the world with startling rapidity, altering the shape of all man's activities, shifting the centres of gravity of power, re-orientating social and individual lives and, as it proceeds, maybe calling for a completely new revelation of man's place in the Universe. We live on the very edge of that revelation in thought and action. Now, at its advent, its nature and character are but darkly understood, its future direction unknown, its seas uncharted. But no man can be unaware of it, even at this stage, and there are features of it which are certain to bring vast changes in the education, outlook and practice of every medical man.

It is not my purpose to traverse them all, even were I equipped to do so. Let us, however, consider one of the most obvious, and certainly one of the most interesting features of the new scientific era, the changing industrial scene as automation and atomic power are projected into the means of production.

Dr. H. Grant-Whyte

* Delivered at the adjourned Annual Meeting of the Association at the South African Medical Congress, Durban, on 16 September 1957.

For many years medical science has directed some of its energies to industrial diseases, of the mind as well as of the body. Man's dependence on work, industrial labour, the sweat of his brow, has had a sort of biblical inevitability, a finality of permanence, around which man has organized all his individual, social and political life. The almost biblically-ordained finality of this order is on the point of disappearing. Yesterday a man was paid to work. The size of our revolution is that tomorrow he will be paid *not* to work. The virtues and compensations of work, daily work, are fast disappearing, and with them will disappear some of the ills of body and mind which are the accompaniments of industrial labour. Already, in some countries the working week and the working day have been so amputated that even now it is possible to foresee a time when the medical problems of men *not at work* will be as important as the industrial problems of men *at work*.

You may have noticed that I have not used the word *leisure* in this context. This word seems to convey a sense of contentment and well-being, but as medical men we are already aware of the ills that beset many people who are leisured in the social and financial sense, some of which ills are the result of being *not at work*, of having *no work*, and of not enjoying the rewarding compensations of work.

It may well be, of course, that the changes of which I speak will come upon us gradually, allowing time for man to adapt himself and his political, industrial and social order to the new conditions. It is true that all scientific progress reveals an ebb and flow, periods of startling advance followed by periods of quiescence, allowing man to catch up with himself, as it were. It seems to be a feature of nature that it shall be so. But what is also true is that the periods of intense scientific activity are occurring at shorter intervals, calling for more rapid adaptation in every department of life, an adaptation which many people fail to make, with results that appear as individual ills of mind and body, and social ills demanding correction and treatment.

I confess I sometimes feel that in the physical sciences and their application, and the early prospect of even greater advances, man may be overreaching himself; and there are many people today, older people, who would advise caution, whisper 'Hamba kahle', go slow, go carefully. But science doesn't work like that, and medical science cannot afford to do so. But I do ask myself quite deliberately whether the sciences—the physical, the chemical sciences, the social sciences, medical science—are moving with the same momentum, the same speed. A striking aspect of modern scientific thought and research, as I see it, is the movement towards a recognition of the truth that all science is one. In the old days, the basic sciences were each treated almost *in vacuo*, as separate studies. Today, physics, the study of the properties of matter, the new physics, has embraced almost all the rest, including a large part, though not all as yet, of medical science.

MEDICINE AND SCIENCE

And in considering the role of medical science in the new world before us, we can pay tribute to the enormous contribution the other sciences have made and are likely to make in the future to the particular science in which we are engaged; noting at the same time that part of the science of medicine which is peculiar and exclusively its own. Today we could not do without the modern tools of medicine, the products of research in other sciences, from radiology to radio-isotopes, and in the future we shall draw from all other sciences, and particularly from atomic physics, the wealth of scientific achievements for application in our work.

There will, however, always be a great difference between medical science and the physical, chemical and engineering sciences. In a sense, medical science includes them all. The great difference is that whereas the sciences, pure and applied, deal with what is embraced by the term Properties of Matter, medical science is the study and care of the individual living man, *Homo sapiens*; and *Homo sapiens* is not only a framework containing all the physical, chemical and engineering sciences, and all the social sciences, but also a mind or, if you like, a soul, with all the creative forces of good within him, and the destroying forces of evil. The medical study of him ranges from his sewerage system to his personality. His ills are not merely functional and organic, matters of engineering and chemistry, and physics. They are psychological and spiritual. *Homo sapiens*, in fact, is a microcosm of the macrocosm of the sciences, not merely of the Universe but of the Cosmos. Indeed, medical science might well, very well, put over its portals the dictum, 'The Proper Study of Mankind is Man'.

And Man is a contrary fellow. He will defy all the laws and the properties of matter. He will go on living when, by all the tests, he should be dead, and he will die when by all the tests he should live. He is at once capable of the greatest sacrifice and courage, and of the harshest cruelty and meanness. Individually a god and a devil by turns, socially an organized insect, an atom in the mass of humanity. Medical science is but one source to which he looks for his welfare and salvation. It is important that it shall be equipped to perform the task awaiting it in the new world.

MEDICINE IN SOUTH AFRICA

For many years we in South Africa have been concerned with the need for the establishment here of advanced schools of surgery and physic, comparable to those of Europe and America, and conferring the degrees and fellowships of the standard and merit we have come to respect elsewhere. These colleges are now in being. They will add much to South Africa's standing in the medical world, and I am happy to think that the first President is with us tonight, my old friend Guy Elliott.

There are other contributions this country and this continent are making and will continue to make to the world's knowledge. Many of our distinguished visitors will no doubt take a piquant and lively interest in our multi-racial society, in Africa's tropical and sub-tropical features, and see something of the work that is being done to meet the peculiar problems that surround us. It has been well said, in this context too, that South Africa is a microcosm of the world, one of the great laboratories of the world, and that it offers in the broad medical sense, as well as in other fields, very special problems for investigation and research.

I have referred, for example, to the manner in which the people of Europe and America are being projected at speed from the pre-atomic age into the atomic age. It is possible, but unlikely, that the passage will be made smoothly, without

ill-effects upon those civilizations. But the African Native, in his millions, is being called on to make a vastly greater change—from a condition of near barbarism, first into an industrial era, and then into an atomic era. The scientific revolution for him is a double one. It is not to be supposed that this can be done without great shocks to African life and well-being.

Admittedly, problems of this size do not touch the average general practitioner, or even the specialist surgeon and physician, though they undoubtedly will do so in the future. I mention them here to call attention to what is, historically, a recent feature of medicine, the World Health Organization and the World Medical Association.

CONCLUSION

Occasionally in recent years I have had the honour and opportunity of attending some of the medical congresses held in other parts of the world, and I have returned only recently from the Congress of the British Medical Association held at Newcastle-on-Tyne. I always leave them with two impressions. The first is how individual a science is medical science, how personal its application, how individual our vocation as medical men, charged with the health and welfare of the patient. I am struck and sometimes awed by the fact that, no matter how vast the changes going on around us, the size of scientific advance, in the final analysis all medicine will continue to depend on the virtues of character and devotion and skill of the general practitioner, the surgeon, the physician, and all those who come into daily contact with the patient.

And the second is how, in the very needs of men all over the world, there has appeared first the world associations of medical men, and out of them, and possibly because of them, a new 'medicine man', a sort of scientist-philosopher-statesman, some of whom are with us tonight; men who, as it were, have ranged the whole scientific world, who have touched most other worlds, and who hold the promise in them of offering to us, and the rest of mankind, a new revelation to serve us as we enter a new era.

Between the two, between the general practitioner on his daily round and nightly calls, and the statesman of medicine, if I may so describe him, lies the range of medicine now, and the role of medicine in the future. I like to think that both at the top and at the bottom, and in between, the organization we need for our tasks shall never organize away the individual character of our vocation, or socialize out of us, as it were, the instinct of service, the spirit of enquiry, the forward-looking mind, and the simple humanity, all of which should, if they do not, invest the science of which we are a part.

To that end, surely, this Medical Congress is devoted, like so many others throughout the world. To this end, all the organization that has gone into it is intended. I, as President, am grateful indeed to all those who have so willingly given of their time, energies, and knowledge, to impress our Congress as a worth-while occasion for the public good.

PRESIDENTSREDE

In hierdie stadium kan dit miskien nie kwaaddoen om saam te vat wat ek reeds gesê het nie. U hoef nie te vrees dat ek alles sal herhaal nie, maar wat ek graag wil herhaal, is my innige spyt dat ons deur omstandighede nie die eer gegun is om sy Eksellensie die Goewerneur-Generaal, Dr. Jansen, by ons teenwoordig te hê nie.

Soos u weet, het hy gedurende sy reis in die buiteland, wat ons almal gehoop het vir hom en haar Eksellensie 'n tyd van rus sou wees, weer in so 'n mate toegegee aan die roepstem van sy plig, dat dit groot eise aan sy kragte gestel het. Ek weet ek praat namens u almal as ek die hoop uitspreek dat, noudat hy terug is in ons sonnige land, hy spoedig sy kragte sal herwin.

Terselfdertyd is dit my aangename plig om by ons te verwelkom Sy Edelagbare Die Administrateur van Natal, Mnr. D. G. Shepstone—'n man wat hom op so baie terreine beywer vir die beswil van ons mense, dat hy ook vir ons 'n ou bekende is. Sy teenwoordigheid hier is vir ons 'n eer en 'n voorreg, en dit gee alreeds aan ons Kongres 'n stempel waarop ons trots is. Aan hom ons hartelike welkom.

Welkom ook aan die uitgelese geselskap besoekers wie se teenwoordigheid onder ons nie net 'n buitengewone eer vir Suid-Afrika se Mediese Kongres is nie, maar ook 'n buitengewone eer vir die land as geheel.

Hulle kom van die setels van geleerdheid van die ou en die nuwe wêreld, en in dié opsig staan hulle nader aan die byna fantastiese veranderinge wat besig is om die wêreld van die wetenskap te oorval.

DIE EEU VAN DIE ATOOM

Die ontdekking dat die atoom nie onverdeelbaar is nie, is besig om die wêreld met 'n ontstellende snelheid te verander, en dit mag uitloop op 'n volkome nuwe onthulling van die mens se plek in die heelal. Geen mens kan hiervan onbewus wees nie, en daar is aspekte hiervan wat geweldige veranderings sal meebring in die opleiding, uitkyk en praktiese optrede van elke medikus.

Ek noem maar een interessante kenmerk van die nuwe wetenskaplike tydperk: die veranderende nywerheidstoneel waar outomasie en atoomkrag in die diens van produksie ingespan word. Die mens het nog altyd sy individuele, sosiale, en politieke lewe rondom sy werk georganiseer.

Hy is betaal *om te werk*. Maar die rewolusie wat voor die deur is, mag daarop uitloop dat hy in die toekoms betaal sal word *om nie te werk nie*, en 'n mens kan 'n tyd voorsien wanneer die mediese probleme van mense wat nie werk nie, net so belangrik sal wees as die industriële probleme van mense wat werk. As medici kry ons alreeds te doen met die kwale van mense wat nie werk nie, wat nie werk het om te doen nie, en gevolglik nie die genot smaak van die beloning van werk nie. Baie mense sal nie die spoedige aanpassing in elke afdeling van die lewe kan maak nie, en die gevolg sal wees individuele kwale van gees en liggaam, en maatskaplike kwale wat behandeling vereis.

DIE MEDISYNE EN DIE WETENSKAP

By 'n oorweging van die rol van die mediese wetenskap in die nuwe wêreld wat op ons wag, moet ons hulde bring aan die geweldige bydrae wat die ander wetenskappe gelewer het en moontlik nog sal lewer tot die besondere wetenskap wat ons bedryf. Tog sal daar altyd 'n groot verskil tussen die mediese wetenskap en die ander wetenskappe wees, want die mediese wetenskap is die studie en sorg van die individuele lewende mens, homosapiens, en sy kwale is nie net funksioneel en organies nie, maar ook sielkundig en geestelik. Die mens is maar 'n snaakse ding: om die beurt 'n God en 'n duiwel, maatskaplik 'n georganiseerde insek, 'n atoom in die groter mensdom. Die mediese wetenskap is maar een oord waarheen hy hom wend vir sy welsyn en sy redding. Dit is dus belangrik dat die mediese wetenskap toegerus sal wees om sy taak in die nuwe wêreld te vervul. Hierdie land en hierdie vasteland het ook sy bydraes om te lewer. Daar is met waarheid gesê, en in hierdie verband, dat Suid-Afrika 'n mikrokosmos van die wêreld se makrokosmos is, een van die wêreld se groot laboratoria, en dat hy sowel in die breë mediese sin, as in ander afdelings, baie spesiale probleme het vir ondersoek en navorsing. Baie van hierdie probleme raak nie die gemiddelde algemene praktisyn nie, maar ek verwys na hulle om die aandag te vestig op 'n onlangse ontwikkeling in die mediese wêreld, die wêreld-gesondheidsorganisasie, en die wêreld mediese vereniging. In die afgelope jare het ek af en toe die eer en die geleentheid gehad om 'n aantal van die kongresse van die wêreld mediese vereniging by te woon. Ek het altyd teruggekom met twee indrukke.

Die eerste is hoe 'n individuele wetenskap die mediese wetenskap is, hoe persoonlik sy toepassing, hoe individueel ons roeping as medici wat belas is met die gesondheid en welsyn van die pasiënt. Hoe geweldig die veranderinge rondom ons ook al is, sal die mediese wetenskap in die laaste instansie tog voortdurend berus op die deugde van karakter, en toewyding, en die bekwaamheid van die algemene praktisyn, die chirurg, die heelkundige, al diegene wat daagliks in aanraking met die pasiënt kom.

Die tweede indruk is hoe daar uit die behoefte van die mens dwarsdeur die wêreld eers die wêreldsverenigings van medici verskyn het, en uit hulle, en moontlik juis vanweë hulle, 'n nuwe soort medikus, 'n soort wetenskaplike-filosoof-staatsman, sommige van wie vanaand by ons is, wat in hulle die belofte inhou vir ons, en die res van die mensdom, van 'n nuwe onthulling wat ons sal bystaan terwyl ons die nuwe tydperk binnetree.

Tussen die twee, tussen die algemene praktisyn en die staatsman van die medisyne, as ek hom so mag noem, lê die hele omvang van die medisyne tans en die rol van die medisyne in die toekoms. Ek wil graag dink dat die organisasie wat ons vir ons taak nodig het nooit die individuele aard van ons roeping sal weg organiseer nie, of die drang tot diens, en die ondersoekende gees, die gees wat vorentoe kyk, die eenvoudige menslikheid as 't ware uit ons sal sosialiseer nie. Want al hierdie dinge behoort, indien hulle dit nie wel is nie, die wetenskap waarvan ons 'n deel is te versier. Aan daardie doelstelling, glo ek, word hierdie Mediese Kongres gewy, net soos so baie ander dwarsdeur die wêreld. Ek, as voorsitter, is inderdaad dankbaar teenoor almal wat so vrywillig van hulle tyd, hulle kragte, en hulle kennis gegee het om hierdie Kongres te bestempel as 'n geleentheid wat die moeite werd is in die openbare belang.

BOOKS RECEIVED : BOEKE ONTVANG

The Anatomy of Congenital Pulmonary Stenosis. By Sir Russell Brock, M.S., F.R.C.S., F.A.C.S. (Hon.). Pp. xiii + 114, with 76 illustrations and diagrams. 30s. net. London: Cassell & Co. Ltd. 1957.

The Care of the Elderly Sick in General Practice. Being the John Matheson Shaw Lecture for the year 1956 delivered in the Hall of the Royal College of Physicians of Edinburgh on 9 November, by W. Ferguson Anderson, M.D., F.R.F.P.S. (Glasg.), M.R.C.P. (Lond.). Adviser to the Western Regional Hospital Board (Scotland) in Diseases of Old Age and Chronic Sickness; Honorary Lecturer in Materia Medica in the University of Glasgow. Pp. 27. Edinburgh: The Royal College of Physicians. 1957.

World Health Organization: Technical Report Series, 1957, *No.* 122. Role of Hospitals in Programmes of Community Health Protection; First Report of the Expert Committee on Organization of Medical Care. 34 pages. Price: 1s. 9d., $0.30, Sw. fr. 1. Available also in French and Spanish.

World Health Organization: Technical Report Series, 1957, *No.* 127. Expert Committee on Biological Standardization, Tenth Report. 35 pages. Price: 1s. 9d., $0.30, Sw. fr. 1. Available also in French and Spanish.

Year Book of Pathology and Clinical Pathology, 1956-1957 Series. Edited by William B. Wartman, B.S., M.D. Pp. 509. 158 Figures. $7.00. Chicago: Year Book Publishers, Inc. 1957.

Lectures on the Scientific Basis of Medicine. Volume Five, 1955-1956. British Postgraduate Medical Federation, University of London. Pp. xii + 473. Illustrations. 45s. net. London: The Athlone Press. 1957.

The Normal Child—Some Problems of the First Five Years and Their Treatment. Second Edition. By Ronald S. Illingworth, M.D., F.R.C.P., D.P.H., D.C.H. Pp. xii + 356. 68 Figures. 33s. London: J. & A. Churchill Ltd. 1957.

Bronchopulmonary Diseases—Basic Aspects, Diagnosis and Treatment. By 142 authors. Edited by Emil A. Naclerio, M.D. Pp. xxi + 956. 719 Illustrations. $24.00. New York: Paul B. Hoeber, Inc.

Oral Cancer and Tumors of the Jaws. A guide for the diagnosis of oral cancer and benign tumors. By George S. Sharp, M.D., F.A.C.S., F.A.C.R. (Ther.), Weldon K. Bullock, M.D., M.Sc. (Path.), John W. Hazlet, D.D.S. Pp. xi + 561. Plates 645. $15.00. New York, Toronto, London: McGraw-Hill Book Company, Inc.

Diagnosis and Treatment of Vascular Disorders. Edited by Saul S. Samuels, M.D. With 17 contributors. Illustrated. Pp. viii + 621. $16.00. Baltimore: The Williams and Wilkins Company. 1957.

Biochemical Disorders in Human Disease. Edited by R. H. S. Thompson, M.A., D.M. and E. J. King, Ph.D., D.Sc., F.R.I.C. 121 Illustrations. Pp. xiv + 843. 90s. London: J. & A. Churchill Ltd. 1957.

A Pocket Obstetrics. Fourth Edition. By Arthur C. H. Bell, M.B., B.S., F.R.C.S., F.R.C.O.G., Hon. M.M.S.A. Pp. 156. Illustrated. 10s. 6d. net. London: J. & A. Churchill Ltd. 1957.

A Practical Handbook of Midwifery and Gynaecology for Students and Practitioners. Fifth Edition. By W. F. T. Haultain, O.B.E., M.C., B.A., M.B., B.Ch., F.R.C.P.Ed., F.R.C.S.Ed., F.R.C.O.G. and Clifford Kennedy, M.B., Ch.B., F.R.C.S.Ed., F.R.C.O.G. Pp. x + 410. 50 Illustrations. 30s. net, postage abroad 1s. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

Progress in Psychotherapy—Volume II. Anxiety and Therapy. Edited by Jules H. Masserman, M.D. and J. L. Moreno, M.D. Pp. viii + 264. $7.50. New York and London: Grune & Stratton. 1957.

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VAN DIE REDAKSIE

NUWE BENADERING TOT AANGEBORE AFWYKINGS

Oor die afgelope paar jaar het die etiologie van aangebore afwykings die belangstelling gaande gemaak, vandat Gregg[1] gerapporteer het dat aangebore katarak (wat altyd toegeskryf was aan oorerflike en genetiese misvorming) in baie gevalle gepaard gaan met en waarskynlik te wyte is aan rubella by die verwagtende moeder. Mediese navorsing het 'n nuwe sfeer van waarneming, met die kans op behandeling en voorkoming, betree.

Twee jaar vóór Gregg se verslag het Wolf, Cowen en Paige[2] feite voorgelê wat 'n protosoön, Toksoplasma, as 'n misvormende faktor blameer. Dit was egter Gregg se verslag wat die aandag van latere navorsers[3] geboei het—hulle was in staat om ook doofheid, hartgebreke en swaksinnigheid toe te skryf aan die baarmoederlike invloed van die rubella-virus.

Die jongste verslae kom van Cambridge, waar Millen en Woollam, uit die Departement Anatomie, die effek van kortisoon plus 'n oormaat van vitamien-A op die swanger rot bestudeer het. Hulle eerste verslag[4] rapporteer 'n groot vermeerdering in die voorkomssyfer van misvorming (36·6 persent) by die kleintjies van rotte wat aan die invloed van hierdie kombinasie onderwerp is, in teenstelling met 'n soortgelyke styging van 7·8 persent by die kleintjies van rotte wat met vitamien-A alleen behandel was en 0 persent by dié van rotte wat met kortisoon alleen behandel was. Cohlan[6] was die eerste om op te merk dat anencephalus, hidrocephalus en exencephalus méér dikwels voorkom by die werpsels van rotte wat gedurende swangerskap 'n oormaat van vitamien-A ontvang het. Die Cambridge-navorsers het hulle dit ten doel gestel om die eksperiment te herhaal, en om ook kortisoon aan die swanger rotte toe te dien.

Hulle tweede referaat[5] is nog meer indrukwekkend as die eerste; onder dieselfde omstandighede is 100 persent van die klein rotjies met 'n gesplete verhemelte gebore ná toediening van kortisoon en 'n oormaat vitamien-A aan hul moeders, terwyl 29·7 persent dieselfde afwyking getoon het op vitamien-A alleen, en nie een op kortisoon alleen nie.

In hul bespreking oor die onderwerp, herinner die skrywers ons daaraan dat Fraser *et al.*[7] opgemerk het dat ‚kortisoon 'n hoë voorkomssyfer van gesplete verhemelte veroorsaak by sekere (stamme van) rotte wat weens genetiese faktore reeds 'n neiging daartoe toon', en hulle besluit op grond van die vorige ondersoek dat ‚die aksie van kortisoon by die aanskakeling van die aksie van vitamien-A eerder 'n algemene dan 'n spesifieke werking is'. Hierdie verslag meld verder dat verskeie ander misvormings by die klein rotjies aangetref is, maar dat ‚slegs dié van die verhemelte in hierdie artikel bespreek word'.

EDITORIAL

NEW OUTLOOK ON CONGENITAL ANOMALIES

The aetiology of congenital deformities has been arousing interest in the last few years ever since the report by Gregg[1] that congenital cataract, which had always been considered a hereditary and genetic malformation, was in many cases associated with—and probably due to rubella in the mother during pregnancy. A new field of observation, with the possibility of therapy and prevention became opened to medical research.

Two years before Gregg's report, Wolf, Cowen and Paige[2] had brought forward evidence which had incriminated a protozoon, Toxoplasma, as a teratogenic agent, but it was Gregg's report that captured the imagination of later workers[3] who were able to ascribe also deafness as well as cardiac and mental deficiency to the intra-uterine influence of the rubella virus.

The most recently recorded papers come from Cambridge, where Millen and Woollam, reporting from the department of Anatomy, studied the effect of cortisone plus hypervitaminosis A on the pregnant rat. Their first paper[4] records a large increase in the incidence of teratomata (36·6%) in the offspring of the rats subjected to this combined influence as opposed to 7·8% in the young of rats treated with vitamin A alone and 0% in those of rats on cortisone alone. The original observation that anencephaly, hydrocephalus and exencephalus occur more frequently in litters from rats which had been overdosed with vitamin A during pregnancy had been made by Cohlan[6] and the Cambridge writers set out to repeat the experiment and, in addition, to give cortisone to the pregnant animals.

Their second paper[5] is even more remarkable; under the same circumstances 100% of the offspring showed a cleft palate on the combined administration of cortisone and overdosage of vitamin A, whereas 29·7% showed the same deformity on vitamin A alone, and none on cortisone alone.

Discussing the subject, the authors recall that Fraser *et al.*[7] recorded 'that cortisone produces an increased incidence of cleft palate in strains of mice in which there is already, for genetic reasons, a liability to the deformity', and conclude from the findings of the previous investigation 'that the action of cortisone in potentiating the action of vitamin A may be a general rather than a specific activity'. It is also indicated in the same paper that various other deformities

Hierdie gegewens is stof vir interessante teorieë. Gesplete verhemelte en haaslip was nog altyd toegeskryf aan 'n oorerflike fout. Dit is beskou as 'n genetiese neiging waarby die wakker klinikus die *formes frustes* kan uitken aan die kort bolip, hoog gewelfde verhemelte en kort bolipgleuf. Kinders met hierdie genetiese trekke is nie noodwendig met 'n haaslip of gesplete verhemelte gebore nie, en die nuwe navorsing dui daarop dat oorproduksie van kortisoon op 'n kritiese stadium van die moeder se swangerskap om die een of ander rede verantwoordelik kan wees vir die feit dat die verskillende gesigsdele nie sluit nie. Daar moet nou voortgegaan word met die navorsing op die gebied van die normale produksie van kortisoon tydens swangerskap en op die faktore wat hierdie produksie enigsins beïnvloed.

In hulle oorsig van die invloed van die rubella-virus op die fetus, het Aycock en Ingalls[8] sekere statistieke voorgelê wat daarop dui dat wanneer die moeder gedurende die eerste 6 weke van haar swangerskap Duitse masels kry, daar 'n hoë voorkomssyfer van katarak by die babas gevind word. As sy dit gedurende die eerste 9 weke kry, kom doofheid voor en, tussen die 5de en 10de week, kan dit hartgebreke en swaksinnigheid veroorsaak. Dit blyk dat die rubella-virus slegs op 'n kritiese stadium van sy ontwikkeling op die vrug inwerk, en moontlik is dit ook die geval met kortisoon. Navorsing moet gedoen word om die juiste tye (wanneer die vrug geaffekteer kan word) vas te stel sodat middels toegedien kan word—om die oorproduksie van kortisoon gedurende hierdie kort tydperk te bestry—aan die verwagtende moeder wie se familiegeskiedenis 'n hoë voorkomssyfer van gesplete verhemelte en haaslip toon.

Volgens die oorlewering, die volkskunde en ouvroue-praatjies sonder tal, word haaslip veroorsaak deur 'n besering of skok wat die verwagtende moeder opdoen, maar aangesien hierdie ‚gesaghebbendes' amper alle aangebore defekte só verklaar, het die mediese beroep nog nooit juis daarvan notisie geneem nie, en dokters was maar altyd geneig om die skouers op te haal. Maar vandag weet ons dat die kortisoonproduksie styg as gevolg van sekere soorte skok en skrik en weens die gevolglike spanning, en dit is heel moontlik dat die dokters verkeerd is en dat die ouvroue tog reg het.

1. Gregg, N. McA. (1941): Trans. Ophthal. Soc. Austral., **3,** 35.
2. Wolf, A., Cowen, D. en Paige, B. (1939): Science, **89,** 226.
3. Swan, C., Tostevin, A. L., Moore, B., Mayo, H. en Barham Black, G. H. (1943): Med. J. Austral., **2,** 201.
4. Millen, J. W. en Woollam, D. H. M. (1957): Brit. Med. J., **2,** 196.
5. Woollam, D. H. M. en Millen, J. W. (1957): *Ibid.*, **2,** 197.
6. Cohlan, S. Q. (1953): Science, **117,** 535.
7. Fraser, F. C., Kalter, H., Walker, B. G. en Fainstat, T. D. (1954): J. Cell. Comp. Physiol., **43,** Suppl., p. 237.
8. Aycock, W. L. en Ingalls, T. H. (1946): Amer. J. Med. Sci., **212,** 366.

were encountered in the young, but that 'only those affecting the palate were considered in the present communication'.

All this leads to very interesting speculations. Cleft palate and harelip have always been considered as being due to some hereditary fault. It was a genetic tendency in which *formes frustes* could be recognized by the astute clinician in the short upper lip, the highly arched palate and the short philtrum. Not every child with these genetic characteristics was born with a harelip or a cleft palate and the new work suggests that for some reason or another, overproduction of cortisone by the mother during a critical stage of the pregnancy may be responsible for the failure of the various facial processes to unite. The field is now open for research on the normal production of cortisone during pregnancy and on the factors responsible for maintaining or altering this production.

In considering the effect of rubella virus on the foetus, Aycock and Ingalls[8] brought forward figures which indicated that rubella contracted in the first 6 weeks of pregnancy led to a high incidence of cataracts in the babies, during the first 9 weeks to deafness and in the 5th-10th week to cardiac and mental deformities. The rubella virus seems to act on the embryo at a critical stage of its development only, and the same may be true of cortisone. Research to establish the exact time relations of this stage is necessary so that drugs to combat the overproduction of cortisone during this short critical period might be given to pregnant women whose family history shows a high incidence of cleft palates and harelips.

Tradition, folklore and innumerable old wives' tales have insisted that harelip is caused by some injury or fright sustained by the expectant mother but, as almost every congenital defect has been ascribed by the same authorities to the identical cause, medical opinion has not paid great respect to this theory and doctors have tended to pooh pooh the old wives' tales. In view of the fact that the production of cortisone is now well known to be increased by certain forms of fright and its resultant stress, it may well be that the doctors will turn out to be wrong, and that the old wives may be right after all.

1. Gregg, N. McA. (1941): Trans. Ophthal. Soc. Austral., **3,** 35.
2. Wolf, A., Cowen, D. and Paige, B. (1939): Science, **89,** 226.
3. Swan, C., Tostevin, A. L., Moore, B., Mayo, H. and Barham Black, G. H. (1943): Med. J. Austral., **2,** 201.
4. Millen, J. W. and Woollam, D. H. M. (1957): Brit. Med. J., **2,** 196.
5. Woollam, D. H. M. and Millen, J. W. (1957): *Ibid.*, **2,** 197.
6. Cohlan, S. Q. (1953): Science, **117,** 535.
7. Fraser, F. C., Kalter, H., Walker, B. G. and Fainstat, T. D. (1954): J. Cell. Comp. Physiol., **43,** Suppl., p. 237.
8. Aycock, W. L. and Ingalls, T. H. (1946): Amer. J. Med. Sci., **212,** 366.

A VARIANT OF RIFT VALLEY FEVER VIRUS

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Two strains of an agent which appears to be either atypically reacting Rift Valley fever (RVF) virus, or a close relative to it, have been isolated from mosquitoes caught in the Lunyo Forest on the Entebbe peninsula. The first strain to be isolated was obtained from a mixed pool of mosquitoes of the genus *Aëdes* and subsequent work has produced evidence that the agent was derived from *Aëdes* (*Banksinella*) *circumluteolus* Theobald. The other strain was obtained about 10 months later from *Aëdes* (*Stegomyia*) *africanus* Theobald caught under similar circumstances in the same area.

Part I of this communication records the isolation of these two strains while Part II describes the results of experiments which were designed to identify them and to show the differences between the locally isolated agent, referred to in this paper as 'Lunyo virus', and classical Rift Valley fever virus.

PART I. ISOLATION

M. B. WEINBREN, M. C. WILLIAMS AND A. J. HADDOW

Detailed descriptions of the routine processes used in this laboratory for attempted virus isolation have recently been given in papers on the isolation of Chikungunya and Zika viruses.[1,2] In view of this, it will suffice here to describe the strains of known RVF virus used in certain experiments:

Pantropic RVF Virus

This is a strain of virus isolated from mosquitoes, *Eretmapodites* spp., caught in Bwamba County, Uganda.[3] It had undergone approximately 160 mouse passages and was used in the form of infective mouse serum.

Neurotropic RVF Virus

This was a strain of RVF virus which had undergone approximately 100 brain to brain passages in mice.

MOSQUITO-CATCHING PROGRAMMES

All the mosquitoes were adult females taken when they alighted on human bait and were caught by a team of trained African youths working under supervision. Each catching period lasted 2½ hours and usually took place during the forenoon, but sometimes in the afternoon and on occasions twice in the same day. The mosquitoes were brought to the laboratory at the end of each catch, in their individual catching tubes. They were then identified and those intended for inoculation were chilled at 4°C to immobilize them and pooled by species. The pooled mosquitoes were then stored at −20°C until suspensions could be made for inoculation into experimental animals.

The first catching programme took place between 26 October and 18 November 1955, during which period 4,130 mosquitoes were inoculated in 29 pools. These mosquitoes are listed below according to the nomenclature used by Edwards.[4]

Anopheles (*Anopheles*) *implexus* Theobald	12
Hodgesia cyptopus Theobald	10
Taeniorhynchus (*Coquillettidia*) *metallicus* Theobald	129
pseudoconopas Theobald	697
maculipennis Theobald	117
fuscopennatus Theobald	212
aurites Theobald	952
(*Mansonioides*) *africanus* Theobald	55
uniformis Theobald	5
Aëdes (*Mucidus*) *nigerrimus* Theobald	1
(*Finlaya*) *ingrami* Edwards	4
(*Stegomyia*) *apicoargenteus* Theobald	13
africanus Theobald	100
(*Aëdimorphus*) *argenteopunctatus* Theobald	4
domesticus Theobald	1
leptolabis Edwards	20
tarsalis Newstead group	4
cumminsi Theobald	11
sp. indet.	1
(*Banksinella*) *circumluteolus* Theobald	1,015+493=1508
Eretmapodites chrysogaster Graham group	21
oedipodius Graham group	125
Culex (*Lutzia*) *tigripes* Grandpré and Charmoy	13
(*Culiciomyia*) *nebulosus* Theobald	1
cinereus Theobald	2
(*Culex*) *annulioris* Theobald	25
univittatus Theobald	5
Culex (*Culex*) *quasiguiarti* Theobald	63
decens Theobald	5
perfidiosus Edwards group	2
guiarti Blanchard	9
moucheti Evans	3
	4,130

In the above list the figures in the right-hand column represent, for all species except *A. circumluteolus*, the total number of mosquitoes caught in 1 afternoon and 19 morning catching periods. During this period the catching team numbered 6, and the figures represent the results of 300 man-hours of catching. The multiple figure for *A. circumluteolus* represents the total as for the catches mentioned above, together with the number of these insects caught during 4 extra morning and 4 extra afternoon sessions. These extra sessions were undertaken when it became apparent that an isolation of virus had been obtained from this species. Towards the middle of November the number of insects taken at each catch fell markedly, probably owing to a seasonal variation, and the programme was brought to an end.

When it was not feasible to inoculate the species processed individually, suspensions were pooled but, though species might be combined in one inoculum, the genera were always kept separate. In those cases in which there was a relatively

large number of mosquitoes of one species to be mixed with a small number of mosquitoes of other species, individual suspensions were made, after which aliquots of each were taken and mixed to form the inoculum. The remainder of each suspension was stored separately, frozen at −20°C to form reference material.

The second catching programme was organized between 9 May and 27 August 1956 in order to attempt virus isolations from *A. africanus*; all other biting insects taken were discarded. During the course of this programme 1,355 *A. africanus* were collected in 80 catching periods and were inoculated as 11 pools. To differentiate these pools from the previously mentioned ones (which were numbered with Arabic numerals), they were designated by Roman numerals. Three strains of virus were isolated during this programme. Pool Lunyo III yielded a strain of Lunyo virus and Pools Lunyo V and VI yielded strains of Zika virus.[2]

THE FIRST ISOLATION OF LUNYO VIRUS

This was obtained from Pool 8, which consisted of equal parts of 2 suspensions made on 5 November 1955 from mosquitoes caught between 31 October and 4 November. The composition of the 2 suspensions is shown below:

Suspension A:	468 *A. circumluteolus* in 10 ml. diluent	
Suspension B:	1 *A. nigerrimus*	in 2·0 ml. diluent
	2 *A. ingrami*	
	1 *A. apicoargenteus*	
	3 *A. argenteopunctatus*	
	4 *A. leptolabis*	
	1 *A. tarsilis* group	
	2 *A. cumminsi*	

The Pool-8 mixture was inoculated into 2 groups of 5 3-day-old mice and 1 group of 5 adult mice. On the 4th post-inoculation day 1 infant mouse was missing from each litter; on the 6th day, in 1 litter 1 infant was missing and another dead, and in the other litter 2 infants were partly eaten. The brains of the dead mouse and of 1 partially eaten infant mouse were salvaged and used to make passages which were successful both before and after the suspension had been passed through a Seitz EK filter pad. The remaining 4 infant mice were sacrificed when sick and their brains were stored in glycerol saline at −20°C. On the 7th day of checking 1 adult mouse was dead; its brain was removed and stored in glycerol saline until 16 February 1956, when it was used to initiate a successful passage series of an agent later shown to be immunologically identical with that obtained from the infant mice. The rest of the adult mice remained well and apparently unaffected by the inoculum.

When the infant mice mentioned above were found to be succumbing to the effects of the inoculum, the stored aliquots of the two mosquito suspensions which were used to make Pool 8 were thawed and inoculated separately into mice. The *A. circumluteolus* suspension was inoculated into 8 litters of 3-day-old mice and 2 groups of adult mice. The other suspension was used to inoculate 2 groups of infant mice and 1 group of adults. None of the mice showed any sign of illness. They were not, however, discarded after the usual observation period, and 6 weeks after they had received their inoculation they were divided into groups, some of which received challenging doses of 3·2 or 32 LD_{50} of 5th-passage Lunyo virus, while the other groups received 63 or 630 LD_{50} of 18th-passage virus. Control groups, consisting of mice of the same age received similar challenges. All the mice which received the larger challenge of 5th-passage virus or any 18th-passage virus died. Of the mice which received the challenge of 3·2 LD_{50} of virus, all those in the control groups died whereas no mice died in 2 groups which had been inoculated as 3-day-old infants with the original *A. circumluteolus* suspension. One of the groups of 5 adult mice which received inocula of this suspension was also challenged with 3·2 LD_{50} of virus, as a result of which 2 died but 3 lived. On 22 November 1955 the remaining 2·8 ml. of the *A. circumluteolus* suspension was thawed and inoculated subcutaneously into rhesus monkey MR 1126. This monkey showed no signs of illness but 3 weeks later its serum neutralized $10^{0·8}$ LD_{50} of Lunyo virus as compared with the pre-inoculation sample, and at 6 weeks after the inoculation it was able to neutralize $10^{1·5}$ LD_{50} of the virus.

The evidence given above concerning the production of a neutralizing substance for Lunyo virus in mice and in monkey MR 1126, after the inoculation of the suspension of *A. circumluteolus*, lends support to our belief that Pool 8 was the origin of the agent although it failed to yield a re-isolation of virus. The fact that, after the original inoculation, the infant mice did not become ill until the 6th day of checking and that only 1 of the adult mice became ill may indicate that there was only a small quantity of active virus in the original suspension, especially as both infant and adult mice, inoculated with material derived from the first mouse groups to be inoculated, were all dead on the 3rd post-inoculation day, thus showing that the agent readily adapts to mice. A further point which was against obtaining a re-isolation is the marked instability of the agent when stored as a frozen suspension of material in bovine plasma albumin (Armour's fraction V, a 0·75% solution in a phosphate-buffered saline pH 7·4. Hereafter in the text this is referred to as BPA). This will be described in more detail in Part II of the communication.

THE SECOND ISOLATION OF LUNYO VIRUS

This was obtained from a suspension of 28 *A. africanus* prepared on 5 June 1956.

This suspension was inoculated into 2 litters of 3-day-old mice, 1 group of adult mice and rhesus monkey No. 1078. On the 7th post-inoculation day 1 of the infant mice died, but the carcase was eaten by the mother and material for passage could not be obtained. None of the other inoculated mice succumbed during a 30-day observation period. Also on the 7th post-inoculation day MR 1078 was found to have a fever of 104°F and was bled. By the following day the monkey's fever had subsided, but it rose again on the 10th post-inoculation day to 104°F, when it was bled again. Isolations of virus were made from both these blood specimens when serum from them was inoculated into mice. On the 8th post-inoculation day it became apparent that no more infant mice of the original groups were likely to be affected by the inoculation, and the stored suspension was thawed and used to inoculate 4 litters of newborn mice in the hope that they would prove more susceptible than 3-day-old mice and would provide an isolation of virus. In 3 of the 4 groups of newborn mice no sign of illness was detected during a 30-day observation period. In the remaining group, however, the mother ate 1 infant on each

of the 5th and 6th post-inoculation days. On the 7th post-inoculation day the mother was found dead and a foster mother was provided for the 3 weak infants which remained. On the 8th post-inoculation day 1 infant had been eaten and on the 9th post-inoculation day the remaining 2 infants were found to be ill. The brains were removed from these ill infant mice and were subsequently found to contain an agent which was shown to be very closely related to the agent isolated from *A. circumluteolus*, if not identical with it, as were the isolates obtained from the serum of monkey 1078.

From this it appears that there was a strain of virus in the *A. africanus* pool which, though it readily infected the rhesus monkey, was only able to induce an apparent infection in very young suckling mice which were rendered more than usually susceptible by reason of intercurrent illness of the mother which led to her death and the consequent starvation and chilling of the infants.

PART II. IDENTIFICATION AND PROPERTIES

M. P. WEINBREN

The clinical picture produced in mice

When inoculated into infant mice Lunyo virus kills them rapidly without the production of unusual symptoms or signs. In adult mice, however, the agent produces a characteristic clinical picture. The mice become markedly hyperactive and when disturbed rush about the cage, occasionally making great leaps so that, when inspecting them in the earlier stages of an infection, the examiner has to exercise great care not to allow a mouse to leap out of the box. This phase is followed by a quieter one, in which the mice sit up with the tail stretched out behind them, rather in the attitude of a kangaroo, while industriously eating any available material. This material is apparently taken without any form of selection, its availability being the most important consideration, and the animals may frequently be seen consuming the bedding or even the carcase of another member of the group which has succumbed more rapidly, though an adequate supply of normal mouse food is within easy reach. Some of the animals appear to suffer from localized areas of irritation or pain; they claw at their own lower jaw and throat, sometimes inflicting severe wounds in the process, while others attack a thigh, or, possibly more often, a fore-limb, when it is not unusual for them to nibble off the entire paw. Mice in more advanced stages of the illness are often seen in convulsions and, in fact, it would appear that the majority of those which die do so while in convulsions. In cases where the animals receive small doses of virus and the survival time is prolonged, paralysis may occur.

Susceptibility of mice to parenteral inoculation of Lunyo virus as isolated

It was noted that Lunyo virus did not affect 5-day-old mice to the degree expected when they were inoculated IP* with 1st-passage material. The results of the experiment described below confirm that, as isolated, Lunyo virus shows a considerable difference in potency when aliquots of the same dilutions are inoculated by the IC† and IP routes.

* IP=intraperitoneal(ly). † IC=intracerebral(ly).

A 10^{-1} Seitz-filtered 4th-passage infant-mouse brain suspension was used to make decimal dilutions down to 10^{-7}: these were used to inoculate groups of 5 adult mice by the IC and IP routes. The results are shown below:

	INTRACEREBRAL ROUTE			INTRAPERITONEAL ROUTE		
Dil.	*Dead*	*Alive*	*Titre*	*Dead*	*Alive*	*Titre*
10^{-1} ..	5	0		2	2	
10^{-2} ..	5	0		3	2	
10^{-3} ..	5	0	$\geq 7{\cdot}3$	4	1	2·7
10^{-4} ..	5	0		0	5	
10^{-5} ..	5	0		0	5	
10^{-6} ..	4	0		0	5	
10^{-7} ..	3	1		0	5	

From the above results it will be seen that there is not only a marked difference in the titre obtained, but also that the mice which do die after IP inocula do not appear to be more likely to do so if given a dose more than 100 times as great as the minimum infecting one.

The occurrence of Lunyo virus in the blood and liver of mice during an intracerebral passage series

In a number of cases mice infected with Lunyo virus show signs of liver damage. It was decided to investigate the relative amounts of virus present in the brain, blood and liver both early and late in the course of an IC passage series, which was likely to have enhanced the neurotropic quality of the agent at the expense of any viscerotropic tendency in the original isolate. The results of parallel titrations of the 3 types of material from 3 different sources of moribund mice are shown below and, from them, it may be seen that at the 24th passage, though no virus is recoverable from the blood, there is still some activity in the liver:

Passage levels ..	4	5	24
Brain	6·0	6·6	6·5
Liver	3·6	4·6	2·6
Blood	1·2	1·8	0

Enhancing the viscerotropic element of Lunyo virus

As isolated, Lunyo virus would appear to be neurotropic rather than viscerotropic though in many, if not all, infected mice there was evidence of some activity in the liver. It was felt that in order to increase the tendency for the agent to grow in the liver, the intraperitoneal route of inoculation should be used, and only livers which showed macroscopic change should be selected to provide material for passage.

A passage series was initiated using 2nd-passage infant-mouse liver which had been stored at −20°C in glycerol saline. This was suspended in BPA and used to inoculate infant mice IP. When they sickened, a number were sacrificed and from them the 3 most markedly affected livers were chosen to make the next passage, which was to both infant and adult mice. When these succumbed all were found to have very pale mottled livers and the clinical picture in the adult mice was less obviously encephalitic. Again the livers showing the greatest degree of change were taken for passage. When the next passage was due it was noticed that the mice showed no encephalitic signs but were dying in a state of prostration within 3 or 4 hours of the first signs of illness. When the passage was made, it was done in the form of two

titrations, one of pooled liver and the other of pooled blood. The liver gave a titre of $10^{5\cdot8}$ and the blood $10^{5\cdot2}$ $LD_{50}/0{\cdot}03$ ml. After this only one more transfer of liver was made. This was from moribund mice in the liver titration; it was in turn set up as a titration, which gave a figure of $10^{7\cdot8}$ $LD_{50}/0{\cdot}03$ ml. From the titration of blood another continuous IP passage series was started in which mouse serum was used as passage material, and after only a few passages the behaviour of the agent was indistinguishable from classical RVF though, as will be shown later, it could be induced to revert to the original pattern very rapidly.

Attempts to neutralize early passage Lunyo virus with known immune sera

On the third post-inoculation day, in the adult group inoculated with Seitz-filtered material derived from the first mouse to succumb to the Pool 8 suspension, 4 mice were dead and 1 was moribund. The dead mice appeared relatively fresh, and their brains were removed and saved for reference material; the moribund mouse was taken for passage. Following the removal of the brains of these mice, the carcases were opened for a routine post-mortem check. One of the dead mice had a pale liver and the moribund mouse had a very pale mottled liver; a specimen of heart blood taken into a capillary tube failed to clot at 20 minutes. In view of these findings, when the routine IC passage of brain material was made to 2 litters of infant and 1 group of adult mice, a spot test was made with known potent RVF antiserum. In the test 10^{-1} and 10^{-4} dilutions of the brain suspension were mixed with equal parts of RVF antiserum and, after incubation of the mixtures (together with controls in which BPA was used in place of immune serum) at 37°C for 1 hour, they were inoculated into litters of 5-day-old mice by the IP route. In the control groups all the mice died at 10^{-1} and 1 of 5 died at 10^{-4}; in the test groups all the mice died at 10^{-1} and all survived at 10^{-4}. The potency of the material was lower than might be expected of a virus which kills adult mice within 72 hours but, as will be shown later, the early-passage material was in a form that does not readily infect mice by the IP route. It was nevertheless felt that, even though the 10^{-4} dilutions gave no definite information, the fact that there was no difference between the 10^{-1} groups was significant, since the RVF immune serum was known to neutralize at least 10^{5} LD_{50} of the homologous virus when tested in adult mice by the IP route.

When it appeared that the test mentioned above was unlikely to prove positive, attention was directed towards Bunyamwera virus, for this was the only agent which was known to give a clinical picture even remotely resembling that of Lunyo virus in adult mice though, in Bunyamwera infection, when a clawing action was seen it was invariably directed towards the nose.

A Seitz-filtered suspension of 4th-passage material was used to set up titrations in BPA and Bunyamwera immune serum. The results showed that there is no inhibition of the action of Lunyo virus by antibodies to Bunyamwera virus.

A more comprehensive test for possible interaction of Lunyo virus and RVF antibodies was then carried out. A suspension of 8th-passage material was prepared and parallel titrations set up, BPA being used as control and a potent human RVF antiserum as test. This antiserum was chosen because it had the highest neutralization index of all the RVF antisera available. In an IP test an aliquot of the same sample had been shown to neutralize more than $10^{5\cdot7}$ LD_{50} of virus. For this test 2 hours' incubation at 37°C was used and the material was inoculated IC into adult mice. The figures obtained for BPA and the test serum were $10^{5\cdot7}$ and $10^{5\cdot3}$ respectively and were taken to indicate that no interaction between virus and antibody was likely to have occurred.

Other known potent immune sera which were tested and found to be devoid of neutralizing power for Lunyo virus were those prepared against Uganda S, Semliki Forest, Pongola,* Spondweni,* Wesselsbron,* West Nile, yellow fever, Bwamba fever, Zika, Chikungunya, Nairobi sheep disease, and Mengo viruses.

A specimen of Lunyo virus was sent to Dr. Max Theiler, of the Rockefeller Foundation laboratories in New York. He reported that though no haemagglutinin had been produced for it, a complement-fixing antigen had been readily obtained. This failed to react with any of the African group-A or group-B antisera, or with antisera to Oriboca, Marituba, sandfly, Bwamba, Bunyamwera, Simbu, blue tongue or horse-sickness viruses. It did, however, react strongly with the homologous antiserum and an antiserum prepared against RVF in Johannesburg by Dr. R. H. Kokernot. In view of these findings, Dr. Theiler felt that there was no doubt that the Lunyo virus is identical or closely related to RVF and, because of this, he decided that no further work with Lunyo virus could be carried out in his laboratory.

Cross neutralization of the viscerotropic strain of Lunyo and RVF

During the period between isolation and the production of the viscerotropic strain of Lunyo, a test had been set up in which specific Lunyo antisera was used against a RVF challenge. This test showed that, even though the Lunyo virus was not apparently affected by RVF antiserum, the reverse was not the case, the Lunyo antisera, whether derived from monkey or rabbit, proving able to neutralize RVF virus in approximately the same degree as homologous antiserum. In view of this finding, and of Dr. Theiler's report from New York, when the viscerotropic strain of Lunyo became available a test was planned in which this agent and classical RVF were used in titrations

TABLE I. RESULTS OF CROSS NEUTRALIZATION TESTS WITH VISCEROTROPIC LUNYO AND RVF VIRUSES (EXPRESSED AS LOGS. VIRUS NEUTRALIZED)

Challenge / *Antiserum*	*RVF IP*	*Lunyo IP*	*RVF IC*	*Lunyo IC*
Titre of virus	$10^{5\cdot5}$	$10^{7\cdot6}$	$10^{5\cdot7}$	$10^{6\cdot4}$
Lunyo monkey ..	6·0	7·0	3·0	1·9
Lunyo rabbit	7·8	7·0		
RVF human	7·5	7·0	3·0	2·2
RVF rabbit	7·8	6·8	3·0	3·0

* These are agents recently isolated in South Africa and antisera to them were provided by Dr. K. C. Smithburn of the Rockefeller Foundation.

of antisera to both, inoculated by both the IC and IP routes. The results of this test are set out in Table I, from which it may be seen that there is a quantitative and reciprocal cross reaction. One may, therefore, assume that in this case the challenging viruses are strains of the same agent.

The degree to which neurotropic and viscerotropic strains of virus may be neutralized

It may be seen in Table I that when inoculated by the IC route a lesser degree of neutralization is obtained for both strains of virus as compared with the IP route. It was at first thought that the relatively low neutralization indices obtained in antisera produced for the original Lunyo isolate, whether in monkeys or rabbits, might have been due to the fact that the agent is a poor antigen. That this is not the case was shown when these antisera were found to be very potent for RVF. Returning to the data in Table I, it may be seen that, although when the IP route is used the viscerotropic strain of Lunyo virus is neutralized by all the antisera to a degree comparable to that obtained in the titrations against RVF, when the IC route of inoculation is used the antisera are able to neutralize less of the Lunyo virus than of RVF. It is thought that this might be because a proportion of the inoculum immediately resumes the neurotropic character. This theory is supported to some extent by the fact that the mouse groups in the Lunyo IC titrations gave a somewhat longer average survival time than in the RVF IC titrations, and many of them, especially at the lower dilutions, showed the encephalitic syndrome described for the original isolate.

In an attempt to discover whether the phenomenon of 'poor neutralizability' of a neurotropic strain was true for RVF as well as Lunyo, titrations of neuro-adapted RVF virus were set up in various antisera using the IC route in adult mice. The results of these titrations are shown below:

BPA control	$10^{6\cdot6}$
RVF immune rabit serum	$10^{6\cdot4}$
Lunyo immune rabbit serum	$10^{6\cdot6}$

The fact that neither of the rabbit antisera was able to neutralize the neurotropic RVF virus when inoculated by the IC route in adults casts doubt on the identity of the virus used. However, when tested by the IP route in infant mice the following results were obtained:

BPA control	$10^{6\cdot9}$
RVF immune rabbit serum	$10^{2\cdot0}$

The results of this experiment show that, as with Lunyo virus, the neuro-adapted form of RVF is much less readily neutralized by an antiserum than the viscerotropic form. Again as with Lunyo virus, an antiserum prepared in rabbits to the neuro-adapted RVF was able to neutralize less of this strain than of the viscerotropic RVF.

Transmission of Lunyo virus by the bite of a mosquito

Early in 1956 a programme designed to show whether Lunyo virus could be transmitted by the bite of various *Aëdes* spp. was begun in collaboration with Dr. A. Michael Davies.* Shortly after the inception of the work, however, Dr. Davies was taken ill and returned to Israel, after which the programme lapsed, owing to which only 1 species of mosquito, *Aëdes* (*Stegomyia*) *aegypti* (Linnaeus), was used and, though the planned experiments were not all completed, it is of interest to quote certain of the results obtained, because they show that this mosquito is capable of transmitting the virus, whereas attempts to induce it to transmit RVF have not been successful.[5,6]

Adult female *A. aegypti* drawn from the Institute insectary were allowed to bite a mouse which was moribund after an intraperitoneal inoculation of Lunyo virus that had undergone 1 IC brain passage and 5 consecutive IP passages of liver. The feeding of the mosquitoes was carried out by placing the mouse, which had been immobilized by the intraperitoneal inoculation of 0·15 ml. of a 2·5% solution of bromethol in saline, into the Barraud cage in which the mosquitoes were kept. After an elapsed time of 1 hour the mouse was removed and sacrificed, and titrations were made of blood, liver and brain; these respectively gave titres of $10^{6\cdot7}$, $10^{5\cdot4}$ and $10^{3\cdot8}$ mouse LD_{50}/0·03 ml. inoculum.

After the feed, all the mosquitoes which contained blood were transferred from the Barraud cage to individual 3 inch × 1 inch tubes.[7] After 20 days, 25 of the fed mosquitoes, which had, in the meantime, been maintained on a raisin diet, were each given the opportunity of feeding on 5-day-old infant mice. Three infant mice were used, each being identified by marking, and the following record was kept of the number of mosquitoes which probed and the number which actually fed on each mouse:

	Mouse No. 1	*Mouse No.* 2	*Mouse No.* 3
No. mosquitoes probed	5	6	4
No. mosquitoes fed	3	2	5

After the feeding, the 3 infant mice were returned to the box along with the mother and their 2 litter mates, which acted as controls to cover the possibility of death from intercurrent illness.

The 25 mosquitoes were triturated in 2·0 ml. of BPA and the resulting suspension titrated in mice, when a figure of $10^{5\cdot4}$ LD_{50}/0·03 ml. was obtained.

The infant mice were observed 6-hourly for 5 days and then 12-hourly for a further 10 days. During this period the 2 normal controls showed no sign of illness but at 66 hours mouse No. 1 was sick and Nos. 2 and 3 were dead and had been partly eaten, although the brains were saved. Mouse No. 1 gradually recovered and was well when discarded on day 15. Two weeks later, the brains which had been removed from mice Nos. 2 and 3 and stored in glycerol saline were used to set up an IC identification test in adult mice. The results of this test are given below:

Antiserum	*Logs Virus Neutralized*
RVF (human)	1·7
RVF (rabbit)	1·9
Lunyo (monkey)	3·0
Lunyo (rabbit)	2·4

From these results it may be seen that the mice died of a strain of Lunyo virus presumably acquired through the bite of *A. aegypti*. An interesting fact is that, though the mosquitoes were infected by feeding on mice circulating the viscerotropic type of virus, the mice inoculated with suspensions of these mosquitoes, or with material from the infant mice which died following mosquito-bite, all showed the characteristic signs of an infection by the neurotropic type of Lunyo virus. This is borne out by the figures given

* A visiting worker from the Hebrew University, Jerusalem.

for the identification test above, for one would expect the viscerotropic strain to be neutralized at least equally well by antisera to RVF and possibly better by the RVF rabbit (see Table I), whereas the Lunyo antisera were more potent than the RVF ones.

It is worthy of note that an almost identical attempt to effect transmission on the 8th day after the mosquitoes fed on the infective mouse failed, though the suspension subsequently made of the mosquitoes gave a titre of $10^{6\cdot6}$ LD_{50}/0·03 ml. being 1·2 logs higher than in the successful transmission.

Stability of Lunyo virus

The agent loses, on an average, a little more than 1·0 log of potency in the process of lyophilization. If the preparation for drying is made in BPA, then the dried virus undergoes considerable further loss on storage at −20°C. When made up as a suspension in non-immune serum, a dried preparation stores much better. When stored frozen at −20°C, a suspension of brain in BPA loses all potency in a short time. A suspension made in BPA was titrated and found to contain 10^7 LD_{50}/0·03 ml. After lyophilization and storage at −20°C for 18 days, the titre fell to $10^{2\cdot8}$ LD_{50}/0·03 ml. and another portion, which was not lyophilized but was stored frozen for the same period, contained no active virus. A preparation of brain in 100% normal serum titred $10^{6\cdot8}$ on manufacture, and the titre fell to $10^{4\cdot5}$ after lyophilization and storage at − 20°C for 6 weeks.

The viscerotropic adaptation of Lunyo virus made possible the collection of infective mouse serum for storage as stock material. This was considerably more stable than the brain suspensions mentioned above. Serum which gave a titre of $10^{7\cdot0}$ when stored at −20°C dropped to $10^{5\cdot8}$ over a period of 28 days. Thus even when stored under the optimum condition of suspension in 100% serum, the Lunyo virus is less stable than classical RVF, for which Mims[8] reports no loss in potency after storage for 33 days at −20°C.

It was discovered that the original Lunyo isolate was most likely to remain viable after a long period if stored as small pieces of infant mouse brain in 50% glycerol saline.

Lunyo virus in monkeys

Rhesus monkey *Macacus mulatta* Zimmerman No. 1131 was bled and its serum tested for antibodies to Lunyo virus. When the results of this test confirmed that the monkey was non-immune it was inoculated subcutaneously with 1·0 ml. of 10^{-1} Seitz-filtered 5th-passage mouse-brain suspension, a titration of which was set up within a few minutes of the monkey inoculation. This titration gave a figure of $10^{6\cdot0}$ LD_{50}/0·03 ml.; thus the monkey received approximately 3 million mouse LD_{50}. The monkey was bled daily for 10 days and the serum so obtained titrated in adult mice inoculated by the IC Route. The results given by these titrations are shown below:

Hours Post-Inoc.	*Titre as Mouse LD_{50}/1·0 ml. Serum*
24	1 mouse of 5 inoculated with undiluted serum died
48	$10^{5\cdot7}$
72	$10^{5\cdot9}$. Monkey had temperature of 103·8°F
90	$10^{4\cdot5}$
115	No activity from this point onwards.

The only reaction shown by the monkey was a fever of 103·8°F on the afternoon of the 3rd post-inoculation day, at which time it was hyper-excitable. Three weeks after the inoculation MR 1131 was bled and its serum was found to contain neutralizing antibodies to Lunyo virus.

Two grivet monkeys, *Cercopithecus aethiops centralis* Neuman Nos. 1139 and 1140, which had been tested and found non-immune to Lunyo virus, each received 1·0 ml. of 17th-passage mouse-brain suspension that gave a titre of $10^{5\cdot8}$ when inoculated IC into mice. They therefore received a dose of active virus comparable in magnitude to that received by the rhesus monkey, but the virus was derived from stock that had undergone a further 12 mouse passages. These monkeys were bled daily for 14 days and titrations were set up with each specimen obtained. During this period neither monkey showed any sign of a reaction to the inoculum, nor was any virus recovered from their serum. Both monkeys were subsequently shown to have produced neutralizing substances for Lunyo virus.

Histopathology

Samples of brain taken from mice infected with early-passage Lunyo material showed an encephalitis of viral type, the only remarkable feature being the presence in the Cornu Ammonis of intranuclear eosinophilic inclusion-bodies, morphologically indistinguishable from those seen in sections of RVF-infected liver. No inclusions could be found in sections of brain prepared from mice inoculated with either viscerotropic or neurotropic RVF.

It has been shown[9] that suspensions containing a high proportion of complete RVF virus produce many blood lakes but few eosinophilic degenerating hepatic cells similar to the Councilman body of yellow fever. As, however, the proportion of incomplete virus in the suspension increases, the blood lakes become fewer and the Councilman-like bodies more numerous.[9] With the viscerotropic strain of Lunyo virus, even when the liver samples are taken from mice with blood titres as high as $10^{8\cdot5}$ LD_{50}/0·03 ml., which according to Mims[10] are exceedingly unlikely to contain much incomplete virus, the most prominent feature of the histological picture in the liver is the very great quantity of the degenerate eosinophilic material. This is very much greater in amount than could be produced experimentally by the deliberate inoculation of mice with suspensions containing high proportions of incomplete virus. In those instances in which liver damage occurred after the IC inoculation of Lunyo virus as isolated, the histological picture seen in the liver sections was indistinguishable from that produced by the viscerotropic type.

The possible isolation of Lunyo virus from a specimen of human blood

In February 1956 G.O., one of the African mosquito-colony attendants, reported sick with symptoms very suggestive of an infection by RVF. He showed a biphasic fever and virus was isolated from blood taken at both peaks of the fever. The only unusual symptoms he reported were dizziness, disturbances of vision and intense pain in the groin. G.O. has little occasion to enter the laboratory building and, if he should do so, he never enters the wing

containing infective material. It is therefore thought unlikely that his infection could have been accidentally acquired in the laboratory. An important point, however, is that the mice inoculated with G.O.'s blood produced signs of nervous involvement and the histological picture seen in their livers was typical of the Lunyo strain of virus.

We were fortunate in having a control for the above-mentioned case in that, 2 weeks after G.O. became ill, A, one of the sterilizing-room attendants, reported sick with what is almost certainly a laboratory infection of RVF resulting from the handling of glassware which had been employed in an experiment with highly infective RVF serum. The course of A's illness was typical of RVF, as was the behaviour of the mice inoculated with his serum.

Serum from both these men was shown to neutralize not only strains of Lunyo and RVF viruses but also the neuro-adapted RVF which, as mentioned earlier, could not be neutralized in an adult mouse IC test by antisera to either Lunyo or RVF.

Haemagglutination by Lunyo virus

Mims and Mason[11] report that RVF serum containing $10^{6\cdot7}$ $LD_{50}/0\cdot5$ ml., in the absence of much incomplete virus, constituted 1 haemagglutinating unit, and also that the infective and haemagglutinating particles are probably identical. In spite of this, Lunyo viscerotropic type of serum containing as much as $10^{9\cdot7}$ $LD_{50}/0\cdot5$ ml. of virus failed to yield a haemagglutinin.

SUMMARY AND DISCUSSION

The isolation of an agent from locally caught mosquitoes (see Part I) and certain of its properties (see Part II) have been described. At first it was thought that the agent might be a strain of Rift Valley fever (RVF) virus because it produced changes in the liver of infected mice. Attempts to neutralize the agent with potent immune sera to RVF failed, as did further attempts with immune sera to 12 other viruses. The virus produces a characteristic pattern of behaviour in adult mice when inoculated intracerebrally; they first become hyperactive and later exhibit an obsessional eating phase during which any available material is swallowed, causing the stomach to become grossly distended; in many instances the mice attack their own throats or limbs, frequently inflicting severe damage; death is usually accompanied by convulsions.

It was found that the agent could not easily infect adult mice when inoculated by the intraperitoneal route, but on selective intraperitoneal passage of liver or blood a viscerotropic type of virus was soon obtained. This readily infected mice by the intraperitoneal route, and cross-neutralization tests carried out with it against classical RVF virus showed a quantitative reciprocal cross, from which it appears that they are different strains of the same agent. This supposition is supported by the findings of Dr. Max Theiler of New York, who carried out complement-fixation tests with the agent. Further differences, however, were found between the Lunyo and RVF strains; some of these are mentioned below.

The Lunyo and RVF strains produce different histological pictures in mice. An isolation was obtained from a human case and, on the basis of the behaviour of the mice inoculated with blood from this patient, and the histological picture produced in them, it is felt that the virus isolated is probably the Lunyo strain. Previous attempts to obtain a transmission of RVF virus by the bite of the mosquito *A. aegypti*[6,7] have failed, though it would appear that this mosquito readily transmits Lunyo virus. However, passage of viscerotropic-adapted Lunyo virus through a mosquito results in its return to the neurotropic type. A similar tendency for classical RVF to adapt very rapidly to a neurotropic variant was seen when it was passed through a local rodent, *Arvicanthis abyssinicus nubilans* Wroughton. This fact was not reported in the paper concerning these experiments, which dealt entirely with the ability of *A. abyssinicus* to circulate RVF and the possibility that it was a natural host in this area.[12]

In view of these facts it is suggested that Lunyo virus is a variant of classical RVF virus, changes in which may have been brought about by the local conditions, and it is not unlikely that, if this is true, other types of variation might occur elsewhere. As an example of this, retinal haemorrhage in man has been reported as a complication of RVF in the Union of South Africa,[13] whereas it has never been reported from other areas. As another possible example, classical RVF virus is extremely stable[8] whereas Lunyo virus and a strain of RVF isolated in Tongaland, South Africa, are unstable under similar conditions of storage.[14]

REFERENCES

1. Weinbren, M. P., Haddow, A. J. and Williams, M. C. (1957): Trans. Roy. Soc. Trop. Med. Hyg. In press.
2. Weinbren, M. P. and Williams, M. C. (1957): *Ibid.* In press.
3. Smithburn, K. C., Haddow, A. J. and Gillett, J. D. (1948): Brit. J. Exp. Path., **29**, 107.
4. Edwards, F. W. (1941): *Mosquitoes of the Ethiopian region III. Culicine adults and pupae.* London: Brit. Mus. Nat. Hist.
5. Smithburn, K. C., Haddow, A. J. and Lumsden, W. H. R. (1949): Brit. J. Exp. Path., **30**, 35.
6. Gillett, J. D. and Cairns, H. J. F. (1954): A.R. E. Afr. Virus Res. Inst., 1953, p. 16. Nairobi: Govt. Printer.
7. Ross, R. W. and Gillett, J. D. (1950): Ann. Trop. Med. Parasit., **44**, 351.
8. Mims, C. A. (1956): Brit. J. Exp. Path., **37**, 99.
9. Weinbren, M. P. and Mims, C. A. (1956): A.R. E.A. Virus Res. Inst., 1955-56, p. 18. Nairobi: Govt. Printer.
10. Mims, C. A. Personal communication.
11. Mims, C. A. and Mason, P. J. (1956): Brit. J. Exp. Path., **37**, 423.
12. Weinbren, M. P. and Mason, P. J. (1957): S. Afr. Med. J., **31**, 427.
13. Freed, I. (1951): S. Afr. Med. J., **25**, 930.
14. Smithburn, K. C. Personal communication.

CONSTRICTION-RING DYSTOCIA*

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With the advent of organized antenatal care and modern methods of combating sepsis, shock and haemorrhage, obstetrical complications have become less hazardous, yet still require all the skill at the command of the accoucheur. We must not rely on all the grand aids at our disposal to the detriment of the fine judgment required in our art. These aids have made Caesarean section—often an obstetrical admission of failure—an operation which can be undertaken under circumstances formerly well known to be lethal or likely to result in a 'near miss'. The 'near miss' is often followed by far-reaching mental and physical sequelae. In order to prevent maternal or foetal conditions from deteriorating to such a degree that all these aids have to be summoned to prevent utter catastrophe, a knowledge of clinical entities, superimposed upon basic clinical principles, is essential. Clear understanding of clinical entities, together with skilful care and judgment, are prerequisites to obtaining the desired effect for mother and child, and for this clear understanding certain basic principles must be understood and known. Failure is a most humiliating experience—especially if the answer is known yet was not considered at the opportune time.

Because of the trouble and the terrible outcome that constriction-ring dystocia often causes, it should be brought to mind whenever there is evidence of an increasing departure from the normal characteristic physiological features of labour.

Constriction-ring dystocia can best be defined as *an abnormal labour which is either due to, or responsible for, the production of a circular, or more or less circular, ridge of uterine muscle protruding into or disturbing the normal contour of the uterine cavity.* The constriction-ring may be found during any stage of labour, most commonly situated at the junction of the upper and lower uterine segments. It may be situated in the upper segment or even in the lower segment. More than one area may be affected simultaneously.

The emphasis of this paper will be on the rings found during the first two stages of labour. The commonest synonym for constriction-ring is contraction-ring, and these terms are to be clearly differentiated from Bandl's ring or the retraction ring, i.e. the normal demarcation between the upper and lower segments. This slight ring-like elevation can be felt within the uterine cavity in some cases (Fig. 1).

Very little is known about constriction-rings, but for purposes of clarity it may be stated that they tend to fall into 2 main groups, as follows:

1. The ring may be just a localized circular area of spasm of the uterine musculature (Fig. 2). It is readily conceivable that by some mechanism (possibly the administration of a drug) this spastic ring may be actively relaxed so that normal uterine action ensues. It may presumably also be prevented by good antenatal care, by paying attention to the psychical (tranquillity) aspect. This type of ring has been referred to by Rudolph[1] as 'reversible'.

2. A localized circular ridge of uterine muscle may be found projecting into the uterine cavity entombing the whole or portion of the foetus behind its grip. The uterine muscle in the affected region is thick (Fig. 3). The upper segment can be seen thinned out from the fundus towards the ring—as the lower segment also may be. Rudolph has classified this group as 'irreversible'. This ring seems to be part and

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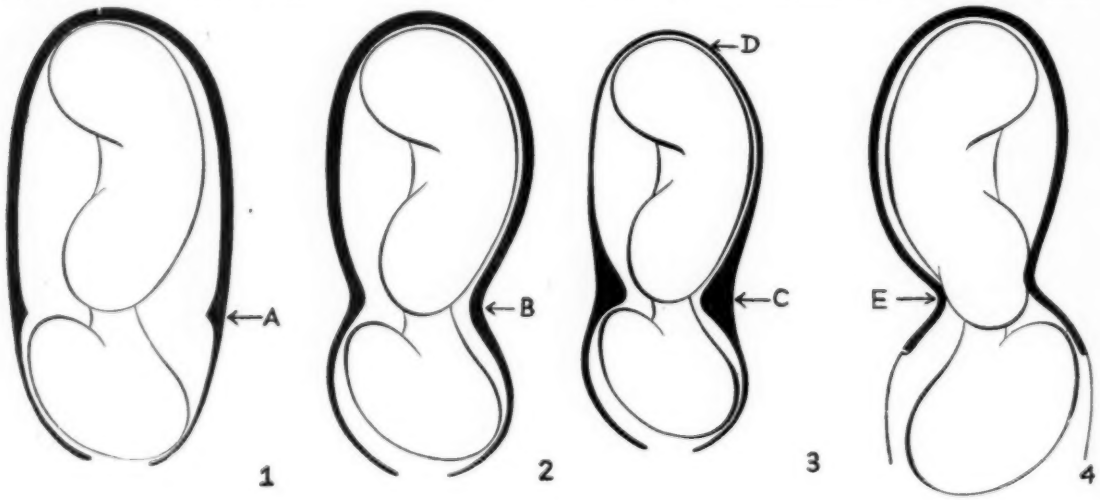
Fig. 1. A=Bandl's or retraction-ring.
Fig. 2. B=Constriction-ring around baby's neck.
Fig. 3. Case 1. C=Irreversible constriction-ring. D=thin upper segment.
Fig. 4. Case 3. E=Constriction-ring around anterior shoulder.



parcel of a grossly abnormal type of uterine action; its correction would therefore conceivably require a complete readjustment of uterine action. This type of ring has been shown by Miles Phillips[2] to persist even after the death of a patient. In the case described by Dauvillier[3] the ring was so tight that it produced oedema of the trapped foetal leg.

One of my patients (F.R.) was gravely ill on admission to hospital as an emergency. She had been in painful labour for 3 days. Vaginal examination confirmed the diagnosis of a tight constriction-ring. After resuscitative measures, Caesarean section was performed. At operation a thick steel-like ring had to be incised in order to release the foetus. The patient died 4 days later of pyelonephritis, and post-mortem examination revealed that the area occupied by the constriction-ring was still readily visible, despite the fact that the uterus had had ample time for complete physiological recovery.

In order to demonstrate certain features of constriction-ring dystocia, summaries of a few pertinent cases will be given. Thereafter an attempt will be made to weave a story through this structure.

CASE SUMMARIES

Case 1. F.A., a 42-year-old 14-gravida, was admitted after being in labour for 40 hours. She had not received antenatal care. No previous labour had lasted for more than 12 hours. Examination revealed a R.O.P. position. The os was 3 fingers dilated and loosely applied to the vertex, which was about 1 cm. above the ischial spines. There was slight caput formation and no moulding. The foetal head remained stationary in the pelvis during contractions, which were abnormally painful. Caesarean section revealed a thick ring around the neck of the foetus. The lower segment was thicker, and the upper segment thinner, than normal (Fig. 3). The live child weighed 8 lb. 3½ oz.

Case 2. An 18-year-old primigravida (M.C.B.) was admitted in labour at term. Antenatally the foetal head had not engaged. Pelvic assessment, clinical and radiological, revealed a largish baby but no cephalo-pelvic disproportion. The patient complained of irregular pains which were mostly situated in the back and just proximal to the symphysis pubis. After 19 hours of labour the os was 1 finger dilated. Examination after a further 10 hours of inefficient labour revealed a 2 finger dilated, poorly effaced, poorly applied cervix. Hyoscine compound B was given, together with intravenous dextrose. When 72 hours in labour—with good rests—neither caput nor moulding were found on examination. The head did not descend during a contraction. Abdominal examination revealed a steep depression between the upper and lower segments. At Caesarean section the picture demonstrated in Fig. 2 was found. The live baby weighed 9 lb. 6½ oz.

Case 3. A 42-year-old 8-gravida (Mrs. v.Z.) was an antenatal case suffering from essential hypertension. Her blood pressure ranged between 150/100 mm. Hg. and 170/100. There was no albuminuria. Labour was induced when she was 35 weeks pregnant because of the steadily rising blood pressure. The method employed was hot bath, castor oil, enema and high puncture of the membranes followed by a pitocin drip. Contractions were poor throughout labour. With the appearance of the head at the vulva and a delayed second stage, forceps were applied, to no avail. The instruments were removed and further vaginal examination revealed a constriction-ring around the foetal posterior shoulder, chest and anterior arm; the anterior shoulder was through the ring (Fig. 4). Amyl nitrite was administered, the ring softened and the baby was extracted. When the placenta was removed manually a ring was still encountered.

Case 4. A.S., a 26-year-old primigravida 36 weeks pregnant, was admitted as an emergency case. She had been in labour for 40 hours when her doctor applied forceps but failed to extract the foetus. On admission maternal and foetal conditions were satisfactory. The vertex presented in the L.O.A. position. The os was fully dilated, there was neither caput nor moulding. A constriction-ring was felt around the neck of the foetus. Deep chloroform anaesthesia and amyl nitrite had no effect on the ring. When Caesarean section was performed the ring was found to be more marked on the *left* side (Fig. 6). The live baby weighed 5 lb. 4 oz.

Case 5. Mrs. P., a 26-year-old primigravida, was 3 weeks overdue. She was in a poor, indefinite type of labour with an L.O.P. for about 140 hours. Her main complaint was a continuous pain on the *right* side which was much aggravated when her bladder filled. At no time was there any caput or moulding. The cervix remained thick and poorly applied. At Caesarean section the ring was found to be thick and marked on the right side and present only to a minor degree on the left side (Fig. 6). The live baby weighed 8 lb. 12 oz. The story and findings in this patient are not unlike those described by Allen.[9]

Cases 4 and 5 may well indicate that the reversible type of ring was gradually progressing to the thick steel-like irreversible one. On the other hand, they may be examples of unilateral congenital uterine abnormalities, but of patterns different from those described by Palmer.[4] The abnormality in these cases might be one of function. The rings described up to this point have all been situated at or near the junction of the upper and lower uterine segments. Case 6, which follows, is of interest as demonstrating the occurrence of a constriction-ring in the upper segment (Fig. 7).

Case 6. Mrs. C., a 37-year-old 5-gravida, was admitted as an emergency case, having been in labour for 12 hours, bearing down for 1½ hours. Her previous labours had been normal. Examination revealed a tired patient with a thick oedematous cervix just visible through the vulva. Morphine and intravenous dextrose were immediately prescribed. Four hours after admission the nurse reported that a deep depression had appeared in the patient's umbilical region. On examination a deep sulcus in the uterus at the level of the umbilicus was both seen and felt. The cervix was still 3½ fingers dilated and poorly applied to the vertex (R.O.P.) which receded with each contraction. A classical Caesarean section was performed in order to sever the ring and deliver the live 7 lb. 9½ oz. baby.

Whatever the aetiology of the ring in case 6 might be, it undoubtedly developed and did not respond to anaesthesia. It had to be severed to free the baby and it was situated in the upper segment. Case 7, which follows, is of great interest because the patient presented with a constriction-ring in the lower segment and another at the junction of the upper and lower segments (Fig. 8).

Case 7. E.S., a 26-year-old primigravida, attended the antenatal clinic regularly from 25 weeks gestation onwards. No abnormality was detected at any stage of pregnancy. She was admitted to hospital with the story that her membranes had ruptured about 6 days previously and that she had been in labour for about 17 hours. Examination showed normal temperature, pulse and respiratory rates; L.O.A. position with the head engaged. The foetal heart was not heard. The contractions were regular. She experienced severe pain during contractions and a persistent lower abdominal pain during uterine diastole. Vaginal examination revealed a loosely applied 4 finger dilated cervix with a constriction-ring below the vertex (at or just above the level of the internal os). It was thought that this os was a loose one, really fully dilated. In the theatre, under general anaesthesia, she was examined once again. The constriction-ring below the head of the foetus had relaxed completely. In feeling around the baby's neck a further vice-like constriction-ring was felt. At operation the ring had to be severed in order to release the 7 lb. 10 oz. macerated foetus.

Case 8. The next case is included for the sake of completeness. It was published in 1947.[5] A 27-year-old 4-gravida was admitted as an emergency case. She was at term, with a story that foetal movements had ceased and that the labour pains were poor and irregular. At odd intervals blood escaped from her vagina. Having had rigors and being too ill to get out of bed, she sent for a doctor who had her admitted to hospital. She was very ill, and there was gas in the pregnant uterus, which did not contract, even when pituitrin was injected. The cervix was soft and 2 fingers dilated.

A rigid channel, admitting one finger only, had to be traversed before the head could be felt. Hysterectomy revealed a gaseous distended uterus with a thick ring distal to the foetal head. The diagnosis of angular pregnancy at term complicated by constriction-ring was made, that is to say, the constriction-ring was situated between the angular pregnancy and the rest of the uterus.

GENERAL

The symptoms and signs of constriction-ring dystocia can be summarized as follows:

Multigravidae sometimes volunteer the information that from the start the pains have differed from those of previous labours. Often they complain of rectal pain and pressure. The contractions may be infrequent, irregular and colicky. Pain is experienced before the onset of contractions and lasts until after their cessation. A ring-like depression may be felt abdominally. Often a circular upper segment and a cylindrical lower segment may be seen and felt (Fig. 9).

During diastole it may be possible to push the head into the pelvis, whereas systole tends to raise the head out of, but hardly ever squeezes it into, the pelvis. Vaginal examination often reveals the absence of caput and moulding, a poorly applied cervix, and a recession or failure of descent of the head during a contraction. The rationale of all these findings is obvious, viz. no outward thrust on the part of the uterus, The diagnosis is only positively made when the ring is felt vaginally or seen when a Caesarean section is done.

Treatment may be outlined as follows:

Conservative treatment consists of adequate sedation, antibiotic cover, and resuscitative measures, together with the administration of relaxants. Rucker[6] has had good results with Adrenalin. Should there be no response to conservative treatment, or should there be added complications (e.g. hypertensive disease, questionable disproportion, etc.), *Caesarean section* is the safest procedure. Any other form of treatment carries too high a foetal mortality rate and severe maternal risks.

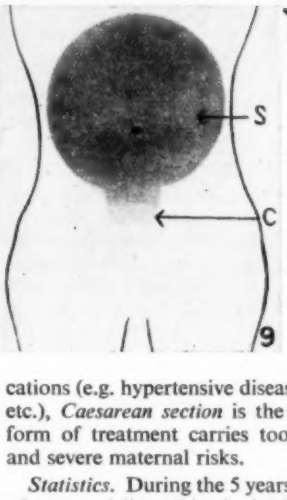


Fig. 9. S=sphere. C=cylinder.

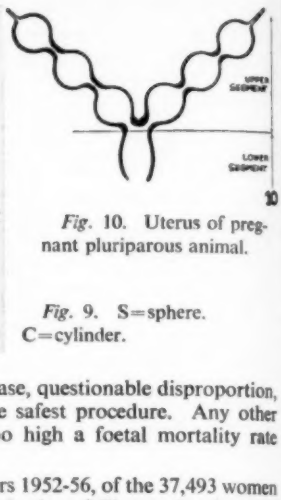


Fig. 10. Uterus of pregnant pluriparous animal.

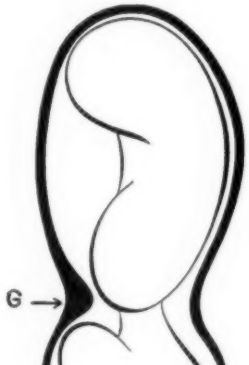
Statistics. During the 5 years 1952-56, of the 37,493 women who were delivered in the institutions falling under the aegis of the University of Cape Town, 56 presented with the obstetrical complication of constriction-ring. Of the babies 16 were stillborn. There was 1 neonatal death. Sixteen patients were primigravidae and 40 multigravidae. Of the latter, 16 had given birth to 7 or more babies. Immunity to constriction-ring dystocia is therefore not conferred by multiparity.

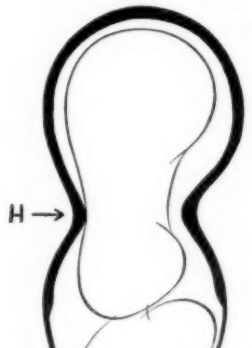
DISCUSSION

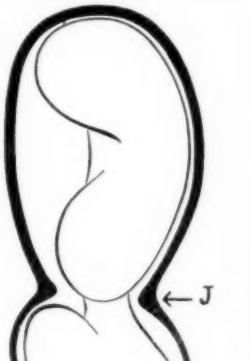
Constriction-rings are to be found in the uteri of pluriparous animals. Tribute must be paid to Rudolph and Ivy[1, 2] for their work on labour in pluriparous animals, e.g. the bitch. In this animal the pups are contained in segments in the two uterine horns, which represent the upper uterine segment in the human female (Fig. 10).

Physiological constriction-rings form (*a*) between the one horn and the lower segment, whilst the other is emptying, and (*b*) in between the young to prevent locking and impaction. These rings, therefore, are situated (*a*) at the junction of the

Fig. 5. a Case 4. F=Constriction-ring, more marked on left side.
Fig. 6. Case 5. G=Constriction-ring, much more marked on right side.
Fig. 7. Case 6. H=Constriction-ring around baby's waist. I=3½-finger dilated oedematous cervix.
Fig. 8. Case 7. J=Constriction-ring around neck. K=Constriction-ring proximal to largest cephalic diameter. L=4½-finger dilated cervix.







upper and lower segments and (*b*) in the upper segment. In the pregnant monkey Ivy *et al.*[8] have shown that stimulation of the lower segment produces a reversal of the normal uterine contraction wave.

Definite conclusions cannot be drawn from these facts; through these facts it has, however, been suggested that under certain conditions (possibly 'stress') the uterine labour mechanism shows atavistic tendencies and that under this influence rings may be formed at the junction of the upper and lower uterine segment, corresponding to the junction of the uterus with its horns in pluriparous animals (cases 1, 2 and 3). These may even be unilaterally situated, as is demonstrated by cases 4 and 5 (Figs. 5 and 6). Rings may also be found in the upper segment, possibly corresponding to the physiological rings found in uterine horns (cases 6 and 8).

The physiology of uterine action is not fully understood. It cannot be said to be completely under psychological control; anyone who has seen a number of deliveries is aware of the fact that some patients who are in dire fear of labour have relatively straightforward deliveries. On the other hand, apparently placid individuals may suffer from incoordinate uterine action. Constriction-ring dystocia is found in primigravidae, multiparae and multi-multiparae and, as has been pointed out, may be found for the first time in a patient giving birth to her 14th baby. Early rupture of the membranes may occur in any normal or abnormal labour, as may also abnormal presentations. Intra-uterine manipulations and oxytocic drugs may predispose to ring formation, but they are not the sole aetiological factors. Labour is often induced by means of rupture of the membranes and the use of oxytocic drugs; yet it is an absolute rarity to find a constriction-ring dystocia in these cases. The uterus belongs to the system of hollow muscular organs. Because of the time factor and the foetal and maternal complications involved, an abnormal reaction on the part of the uterus is a much more serious event than is a spastic colon for example. A patient suffering from spastic colon complains of inability to have a proper motion. The colon becomes 'knobbly' around balls of faeces not unlike the colon of the goat or sheep. However, this is not considered an atavistic tendency on the part of the gut. The time factor in spastic colon is not of great importance, whereas in labour it is of the utmost significance. Atavism, therefore cannot be loosely accepted as an aetiological factor. However, it is possible that the sites of constriction-ring formation have evolutionary significance.

Physical interference acting in an individual with an abnormal psychical background, as is found in most cases of spastic colon (i.e. a tenseness produced by some minor degree of psychological imbalance), may be at the root of the evil. The psychological explanation is an excellent shelf upon which to place unexplained phenomena and so unburden the mind of difficulties. It is not known whether a patient who, having had proper antenatal care, has *inter alia* been taught self-reliance, has developed constriction-ring dystocia. Not until the basic physiology of the different varieties of smooth muscle is understood will this closed door be opened.

SUMMARY

1. The constriction-rings of labour are defined.

2. Case histories of a few patients are briefly given in order to demonstrate the different situations in which constriction-rings have been encountered.

3. The symptoms, signs and treatment of constriction-ring dystocia are briefly outlined.

4. A few aetiological features are considered. Unfortunately, firm conclusions cannot be drawn from either argument or observation.

REFERENCES

1. Rudolph, L. (1935): J. Obstet. Gynaec. Brit. Emp., **42**, 993.
2. Phillips, M. H. (1934): *Ibid.*, **41**, 497.
3. Dauvillier, P. W. (1951): Ned. T. Geneesk., **95**, 535.
4. Palmer, A. C. (1951): Proc. Roy. Soc. Med., **44**, 867.
5. Louw, J. T. (1947): J. Obstet. Gynaec. Brit. Emp., **54**, 477.
6. Rucker, P. (1946): Amer. J. Obstet. Gynec., **52**, 984.
7. Rudolph, L. and Ivy, A. C. (1930): *Ibid.*, **19**, 317.
8. Ivy, A. C., Hartman, C. G. and Koff, A. (1931): *Ibid.*, **22**, 388.
9. Allen, L. A. (1952): S. Afr. Med. J., **26**, 398.

PROBLEMS OF BALLISTOCARDIOGRAPHY

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Nearly 80 years ago, in 1877, Gordon published a paper on 'Certain Molar Movements of the Human Body Produced by the Circulation of the Blood',[1] in which he described the first low-frequency ballistocardiograph. In spite of the vast amount of work reported in the intervening years, it is still evident that the ballistocardiograph has not achieved the accepted status of the much younger phonocardiograph and electrocardiograph.

From an empirical approach much has been achieved. The amplitude of the ballistocardiogram has been shown to have some relationship to cardiac output, and fairly characteristic ballistocardiographic patterns show correlation with certain pathological conditions. However, there is as yet no clear understanding of the causal relationship between specific cardiovascular events and corresponding ballistic waves, and ballistocardiography is still far from being a precise science. Dock[2] has concluded that 'the ballistocardiographic record is read much as one reads the facies of a patient; it is not subjected to quantitative analysis but is intuitively compared with the normal range for young adults'.

It is not the intention here to review the present status of ballistocardiography, which has been admirably done elsewhere, [3-5] nor to contrast current methods and techniques,[5] but to consider some of the great difficulties inherent in the precise interpretation of ballistocardiographic events and to

suggest that these may have been unnecessarily increased by the experimental methods employed.

The forces set up by cardiac ejection have been compared to the recoil of a gun. If the gun were suspended in space and fired it would be displaced in the opposite direction to the bullet. Its recoil would depend on 3 variables, the mass of the gun (heart weight), the mass of the bullet (stroke volume) and the force of the explosive (ejection force). However, the body is a self-contained system and the bullet does not fly off into space. A better analogy, therefore, would be an iron box with the gun attached to the floor. Were the base placed on a ballistocardiograph platform so that its movements could be recorded, there would again be a displacement of the gun and box in the opposite direction to the bullet, the movement, however, being rapidly checked and reversed when the bullet hit the wall of the box. Thus the *distance displaced* and the *velocity achieved* by the box during the initial recoil would also depend on a most important 4th variable, the time taken for the bullet to strike the opposite wall (determined by its speed and the distance to be travelled.)

It is already apparent that the initial deflection of the ballistocardiogram is subject to 4 major variables and cannot be expected to correlate with any one of these alone. However, the above analogue is greatly over-simplified. For it to simulate the body more closely the gun (heart) should not be bolted to the box (skeleton), but should be attached by elastic and movable supports (fascia, liver, lungs etc.), which to an unknown and varying extent would absorb the shock transmitted and produce after-vibrations due to their being themselves set in motion. In addition, the gun should be set at an unknown and variable angle to the sides of the box, the forces set up being therefore only partially recorded in the long axis (head-foot) of the box. Finally, the stroke output does not remain as a single bolus, but splits up into the branches of the aorta. In terms of our simile the bullet must be represented as fragmenting in different directions. The forces set up by deceleration of blood in the aorta, hands, legs, carotid and pulmonary vessels partially summate in time and are variable and hard to isolate.

The above is an attempt to represent some of the complexities of BCG interpretation in terms of a mechanical analogue. No attempt is made to cover any but the early systolic events, but it is already clear that the stroke volume and heart force are only two of many factors influencing the magnitude of the ballistocardiographic deflection. However, in most ballistocardiographic systems there is one further very considerable complicating factor.

To return to the mechanical analogue, let us suppose that the box was not standing firmly on the BCG table, but instead on a sorbo-rubber mattress (representing the soft, subcutaneous tissue padding of the body) between box and bed. The resultant BCG record would now be further distorted in 3 ways: (1) High-frequency vibrations would be greatly attenuated by this shock absorber; (2) phase shifts would appear in those vibrations that were transmitted; and (3) a series of after-vibrations would be set up which would largely obscure the later intrinsic vibrations arising in the box system.[6-9]

This is precisely what happens when ballistocardiograms are recorded by most current methods. In all those ballistocardiographs in which restraint is applied to free body movement distortion takes place in the final record; that is to say, there is phase shift and attenuation of high-frequency vibrations, and the after-oscillations of the body on its tissues are superimposed on the oscillations due to cardiovascular events.

Restraint of body movement may be applied by stiff springs (e.g. Starr's table) or by placing the body on a rigid surface (Dock's method). Even with a low frequency suspended bed without springs the very mass of the bed may of itself offer a resistance to the relatively high frequency oscillations consequent on the heart beat. In Fig. 1 each trace is the result of striking a normal subject a sharp blow on the head. The sensitivity has been sufficiently attenuated to obliterate oscillations of cardiovascular origin. The bottom trace (DF) is the record obtained with a Dock ballistocardiogram, with the subject lying supine on the floor, and represents the displacement produced by the blow and the after-oscillations of the body on its tissues. The upper two traces on the left show the acceleration and velocity (A and V) achieved by a 4½ lb. low-frequency unclamped bed[10] when the same subject is struck a similar blow while lying on the bed. It will be seen that after-oscillations are negligible. The upper two traces on the right show the results of a similar experiment with the bed weight artificially increased to 40 lb. The greater amplitude of the initial displacement simply reflects the fact that a harder blow was delivered and is of no significance, but the striking after-oscillations are most evident.

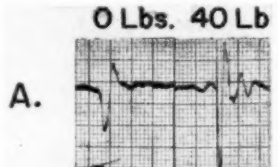

Fig. 1. A series of records obtained by striking an individual a blow on the vertex and procuring records in the following manner: DF = Subject supine on the floor; relative movement of patient on floor recorded with Dock instrument. A and V = Acceleration and velocity records with subject on a Rappaport bed (on the left) and the same bed with its weight artificially increased by 40 lb. (on the right). There is sufficient attenuation to obliterate complexes of cardiovascular origin.

The results of progressive increments in the weight of the bed are evident in Fig. 2. Here the acceleration and velocity (A and V) ballistocardiograms of a normal individual on the same type of bed are compared to the relative movement of the subject on the bed recorded by a Dock instrument (D) and the heart sounds as a reference record (HS). It is apparent that as the bed weight is increased from 0 to 40 lb. there is progressively more movement of the body on the bed (trace D) and progressive distortion of the acceleration and velocity ballistocardiogram.

The principle that a recording system should offer minimal restraint to free body movement was appreciated by Gordon[1] and has been stressed in varying degree by more centre workers.[5-9] The extent to which departure from this principle has impeded progress will become apparent as studies are repeated on non-resisted systems. However, it already seems likely that some of the 'abnormal' patterns observed

Fig. 2. Acceleration (A) and Velocity (V) ballistocardiogram of a normal individual on a Rappaport 4½ lb. bed to which weights have been artificially added in 10 lb. increments up to 40 lb. Trace D records the relative movement between the subject and the bed recorded by a Dock instrument. HS=heart sounds.

in 'normal' subjects may be primarily the result of changes in the body-supporting tissues and it has already been demonstrated that some of these patterns may become normal when the subject is bedded in a firm supporting medium such as putty or sand.[11]

Finally, it is probable that progress in understanding of the ballistocardiogram has not been expedited by the large number of sensing devices that have been employed. These are numerous, and include techniques which record the displacement (movement), velocity (rate of movement), and acceleration (rate of change of velocity) of the body on a rigid support, or of the bed. No attempt should be made to equate the results of these different techniques without a clear concept of their differences. The results of two of these techniques are somewhat comparable, though differing greatly in their methods. In a table of the type described by Starr, with head and foot boards applied, body movement is restricted as far as possible, and that movement which does take place is against stiff springs. The *displacement* of this bed is, therefore, a measure of the *force* applied. In a system with minimal restraint (Rappaport[10]) the sensing device records the *velocity* achieved by the bed, and by electrical differentiation the *acceleration*. When it is remembered that force is the product of mass and acceleration it will be seen that each of these systems is recording a comparable parameter, though the errors introduced by the resisted systems (described above) will cause large differences in the resultant records. Techniques which explore the *displacement* of a body against the resistance of its own tissues or of beds with lesser degrees of resistance are obtaining records which relate to force more closely as the movement is restricted. The resultant records are more or less impure mixtures of force and displacement, and are impossible to represent in the language of simple physics.

In conclusion, it is clear that in spite of some progress on empirical lines there is as yet very little understanding of the aetiological relationship between ballistocardiographic deflections and cardiovascular events. The factors requiring analysis are most complex and some of these have received mention above. However, it seems probable that one of the causes of the slow progress in the interpretation of the ballistocardiogram may be found in the use of systems offering unnecessarily high resistance to body movement and in the number of different sensing devices that have been employed. The fact that distinctive ballistocardiographic changes have already been found to correlate with certain disease states indicates that better understanding of the aetiology of the ballistocardiogram is an eminently worth-while goal. Until, however, this goal has been more nearly attained the ballistocardiograph should remain an instrument of research and its widespread application in clinical medicine would be most premature.

REFERENCES

1. Gordon, J. W. (1877): J. Anat. Physiol., **11,** 353.
2. Dock, W., Mandlebaum, H. and Mandlebaum, R. A. (1951): J. Amer. Med. Assoc., **146,** 1284.
3. Singewald, M. L. (1954): Ann. Intern. Med., **41,** 1124.
4. Scarborough, W. R., Davies, F. W. Jnr., Baker, B. M. Jnr., Mason, R. E. and Singewald, M. L. (1954): Amer. Heart J., **44,** 912.
5. Talbot, S. A. and Harrison, W. K. Jnr. (1955): Circulation: **12,** 577.
6. Rappaport, M. B. (1955): Mod. Conc. Cardiovas. Dis., **24,** 277.
7. *Idem* (1956): Amer. Heart J., **52,** 483.
8. Burger, H. C., Noordergraaf, A. and Verhagen, A. M. W. (1953): *Ibid.*, **46,** 71.
9. Von Wittern, W. (1953): *Ibid.*, **46,** 705.
10. Rappaport, M. B. (1954): Abstr. 2nd Wld. Congr. Cardiol., p. 229. New York: Amer. Heart Assoc.
11. Walker, R. P., Reeves, J. T., Willis, K., Christianson, L., Pierce, R. and Kahn, D. (1953): Amer. Heart J., **46,** 166.

PSITTACOSIS IN JOHANNESBURG

A CASE OF ATYPICAL PNEUMONIA

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Psittacosis is a specific infection of the psittacine or parrot family of birds including parrots, parakeets, cockatoos and budgerigars, and has long been known to be directly transmissible to man. Virus of the psittacine family infects many other non-psittacine species of birds, and canaries, pigeons, turkeys, domestic fowls, ducks, fulmar petrels and finches are known to contract the disease, and may infect man. The term 'ornithosis' is applied to non-psittacine infections. The evidence is that a vast avian reservoir of infection exists in domestic and wild birds.

Very few human cases are known to have been diagnosed in South Africa but the incidence of psittacosis is considerably higher than is realized and, with the increasing popularity of the budgerigar as a domestic pet, outbreaks of the disease can be expected.

The first cases of psittacosis were reported in 1880 and a pandemic originating in infected South American parrots occurred in Europe towards the end of the 19th century. In 1930 the virus responsible for the psittacosis-ornithosis group of infections was identified independently by several investigators. There is a high incidence of the disease in America and in the years 1952-55 the number of cases reported annually to the US Public Health Services varied from 135 to 495. 200 cases attributed to transmission from infected turkeys occurred in the US during 1954; otherwise infections have generally resulted from contact with infected psittacine birds.

The causative agent is one of a group of large filterable viruses of the psittacosis and lymphogranuloma inguinale group, antigenically related on the basis of complement-fixation tests.[1,2] Infected birds, especially newly imported birds, represent the main source of infection. Though usually ill, with listlessness, rough feathers, coryza and nasal discharge or bloody diarrhoea, they may be apparently healthy. Inhalation of dust from cages or aviaries contaminated by infected discharges, inhalation of feathers, and bites by infected birds, are the main source of infection. Hence it is thought that infection is commonly contracted during the cleaning of the bird's cage, as in the case reported here. Transmission from man to man sometimes occurs but is rare.[3-5] Clinical infection is considered to be rare in children and infants but that this may not be so is suggested by Berman *et al.*,[6] who discuss a number of cases occurring in infants in Israel in 1953 and 1954.

The Law

In 1930, after a serious outbreak of psittacosis in Britain with many deaths, a complete ban on the importation of psittacine birds into Britain was imposed. The ban was lifted in December 1951. Many outbreaks and some deaths from the disease have since been reported, and the embargo has been re-imposed. An outbreak of psittacosis, the first in 10 years, occurred in New York shortly after revocation of a law prohibiting the sale, breeding or importation of psittacine birds in New York state and New York city early in 1952.[4] In South Africa, Proclamation No. 211 of 19 May 1931, issued under sections 76 and 84 of the Public Health Act, prohibits the introduction into the Union by land or sea of all birds of the parrot family (psittacidae) and lovebirds and budgerigars (variously classified as Psittacidae or Psittaculidae) except where a permit therefor has first been issued by the Secretary for Public Health, subject to compliance with any conditions or requirements, such as a period in quarantine, stated in such permit. Psittacosis is a 'proclaimed disease' under the Stock Diseases Act 14 of 1911, and in practice permits are issued by the Veterinary Department. Permits are fairly freely issued but for restricted numbers and subject to veterinary certificates and quarantine of birds. Psittacosis, according to notifications, is by no means common in birds in this country.

CASE HISTORY

On 2 October 1956, during a polio epidemic, a European male aged 33 years, a fitter and turner, was admitted to the Johannesburg Fever Hospital with a provisional diagnosis of 'encephalitis', compaining of headache and low back pain for 3 days and cough for 1 day. He awakened on the morning of the commencement of his illness with a generalized throbbing headache felt particularly in the occipital and retro-orbital region and aggravated by moving and coughing. His eyes felt 'as if they would burst out', and he had aching lumbar pain (he had been diagnosed as having influenza), a sore throat, and an irritating non-productive cough which started on the day of admission to hospital, but no chest pain. He complained of difficulty in breathing. Questioning revealed anorexia of 2 days duration with nausea and constipation.

The patient lived in Johannesburg and had not been away for several months. He had been seen by one of us 7 years previously and had then been diagnosed as suffering from Bornholm disease.

On admission his temperature was 100°F and pulse rate 108 per minute. He appeared very ill, with marked irritability and congestion of the face and neck, conjunctival injection, and marked photophobia. The pharynx had a red granular appearance and the tongue was heavily coated. The cardiovascular system was apparently normal. Deep inspiration brought on paroxysms of coughing and occasional rhonchi were heard in the chest. The abdomen was slightly distended and there was tenderness in the right hypochondrium. The liver and spleen were not palpably enlarged. The tendon reflexes were depressed, but no paresis was found and no stiffness of the neck or back. The cranial nerves and fundi were normal.

During the 3 days after admission the temperature ranged between 100 and 103·4°F. He complained of very sore throat and severe headache. The paroxysmal cough persisted and he had one small epistaxis. Scanty pink macules resembling typhoid spots were observed in both flanks. Moist rales were heard in

the left axillary region. A portable X-ray on 6 October 1956 revealed a consolidation in the left mid-zone, and at this stage the patient coughed sputum streaked with bright red blood (Fig. 1).

Laboratory investigation was carried out on admission as follows: Blood: white-cell count 9,000 per c.mm. (polymorphs 85%, lymphocytes 11·5%, monocytes 3·5%) CSF: Clear; polymorphs 3 per c.mm., lymphocytes 4; protein 31 mg.%, sugar 65 mg.%. Blood culture in bile and broth, negative. Rickettsial

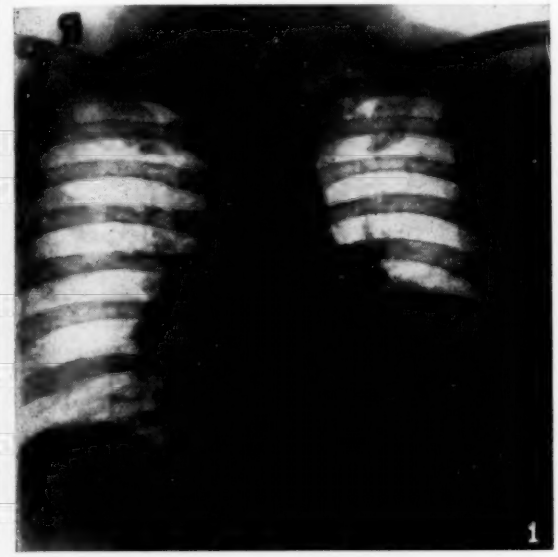

Fig. 1. X-ray of chest on 6 October 1956.

complement-fixation tests, negative. Cold agglutinins not present in the blood. Stool and urine cultures, negative for typhoid. Urine: No albumin, no sugar, bilirubin absent, urobilin +. Blood bilirubin: 0·4 mg.% on admission, 1·2 mg.% 6 days later.

A diagnosis of atypical pneumonia was made and investigation instituted to exclude psittacosis and other causes. On questioning, the patient said he had acquired a budgerigar on the day before his illness, the only contact with any other bird having been 2 weeks previously, when he had helped a friend clean out and build a ladder in the cage of his budgerigar.

The patient remained in hospital and was febrile for 8 days. Penicillin was administered from the 6th to the 11th day in dose of ¼ million units 6-hourly and again in reduced dose from the 13th to the 17th day. After the temperature fell convalescence was uneventful.

A positive psittacosis-lymphogranuloma complement-fixation test was obtained in titre 1 : 10 from blood taken in the first week of illness. One week later the titre had risen to 1 : 100, and 10 days later to 1 : 200, indicating that the illness had in fact been psittacosis.

Enquiry revealed that the budgerigar inhabiting the cage worked on by the patient 2 weeks before his illness had never been well and that a canary had died in the same cage a short time before. Examination of the bird at the Poliomyelitis Research Foundation Laboratories, Johannesburg, revealed the presence of psittacosis virus.

DISCUSSION

Clinical Aspects of Psittacosis. Both the clinical manifestations and the severity of the disease are variable. The incubation period is usually 10 days, with extremes of 4-15 days. An abrupt onset is usual. In mild cases there is severe headache, with photophobia, malaise, fever, and pain in the back and limbs, the picture being that of 'pyrexia of unknown origin' or an influenza-like illness lasting 3 days to a week with uneventful convalescence. In severe cases signs and symptoms of lung involvement occur after a few days of intense headache, profound weakness, prostration and myalgia. Tachypnoea, chest pain (which may be pleural), and cough, either unproductive or with haemoptysis, are symptoms. There may be severe abdominal pain, nausea, vomiting or diarrhoea. Epistaxis and conjunctival injection or subconjunctival haemorrhage occur. There may be neck stiffness, and sore throat is common.

Diagnosis. The most generally applicable test is the demonstration of psittacosis antibody in the patient's serum. Two specimens are required, one in the first week and one about the 21st day. Chemotherapy may delay the appearance of antibodies for 3-6 weeks or even prevent their appearance. The finding of antibody in low titre in the first specimen suggests that the patient has had an infection with a virus of the psittacosis-lymphogranuloma group. A high titre of 1 : 30 or over in the first specimen is suggestive of the disease, and a 4-fold or greater rise in the second specimen is diagnostic. Meyer and Eddie[7] state that examination of repeated specimens for complement-fixing antibodies with a 2- to 4-fold increase in titre plus evidence of pulmonary involvement are sufficient to warrant the diagnosis of psittacosis; also that a titre of 1 : 16 obtained from a patient with clinical manifestations of psittacosis may be considered positive. It is also possible to isolate the virus from the patient's blood in the early days of infection and from the sputum when lung involvement has occurred. The isolation of virus from a bird contact, as in the case reported, gives further evidence of the nature of a suspected infection.

The Carrier State. Although the healthy carrier state is well known in birds, its occurrence in man is probably rare, although Eddie[8] reported a human carrier of psittacosis who harboured the virus for 10 years after a severe acute attack. Patients have spread the disease to nursing contacts and others.

Treatment. Case fatality has decreased from 50% to 2% with antibiotic therapy. Good results have been reported with the use of penicillin, which should be administered in large doses up to 2 million units per 24 hours. Chlortetracycline (Aureomycin) and oxytetracycline (Terramycin) have given equally good and possibly better results; chloramphenicol (Chloromycetin) is less active. Antibiotics are probably virustatic and not virucidal, hence the failure of chlortetracycline to clear up the carrier state in birds.[9]

Résumé. The clinical picture of psittacosis is usually that of an acute disease with an incubation of about 10 days. The symptoms are generally malaise, fever, severe headache and myalgia, and symptoms of nausea, vomiting and photophobia. Often no other abnormal signs are found and after a few days of pyrexia the patient recovers. In severe cases, towards the end of the first week there is evidence of pulmonary consolidation, a cough, usually unproductive, and chest pain which may or may not be pleural in nature, and the spleen may enlarge. The pulse is often relatively slow. Rose spots are noted in a few cases and a scarlatiniform rash has been described. Without chemotherapy the disease carries a high mortality. The symptoms of psittacosis are liable to be confused with those of influenza, 'Q' fever and other forms of atypical pneumonia, typhoid fever, and meningo-encephalitis, especially in cases characterized by

conjunctival injection and photophobia such as leptospiral meningo-encephalitis.

SUMMARY

1. A case of psittacosis is described, presenting with atypical pneumonia.

2. Diagnosis was established by the demonstration of complement-fixing antibodies in diagnostic titre for the psittacosis and lymphogranuloma venereum virus group and the isolation of virus from a budgerigar contact.

3. The clinical features of psittacosis are discussed.

4. Attention is drawn to an increasing incidence of the disease abroad and the suggestion is made that psittacosis is not very uncommon in South Africa.

We wish to thank Dr. J. W. Scott Millar, Medical Officer of Health, Johannesburg, and his department for their cooperation and the staff of the Poliomyelitis Research Foundation for their assistance in the pathological investigations.

REFERENCES

1. Rivers, T. M. (1952): *Viral and Rickettsial Infection in Man*, 2nd ed., p. 440. Philadelphia: J. B. Lippincott Co.
2. Meyer, K. F. and Eddie, B. (1951): Bull. Hyg. (Lond.), **26**, 1.
3. Hansen, P. F. and Sorensen, L. B. (1955): Dan. Med. Bull., **2**, 51.
4. Perlman, L. and Milzer, A. (1954): Arch. Intern. Med., **94**, 82.
5. Singer, E., Sussman, O. and Barnett, J. C. (1956): Amer. J. Med., **20**, 153.
6. Berman, S. *et al.* (1955): Pediatrics, **15**, 752.
7. Meyer, K. F. and Eddie, B. (1947): J. Amer. Med. Assoc., **133**, 822.
8. *Idem* (1951): J. Infect. Dis., **88**, 109.
9. Quan, S. F., Meyer, K. F. and Eddie, B. (1950): *Ibid.*, **86**, 132.

ANNUAL GENERAL MEETING OF MEDICAL ASSOCIATION

The Annual General Meeting of the Medical Association of South Africa was held in the B.E.S.L. Hall, Old Fort Road, Durban, on the morning of 11 September, when Dr. Harry Grant-Whyte was inducted as President of the Association by Dr. J. S. du Toit, the retiring President.

Dr. du Toit referred to the great services which Dr. Grant-Whyte had rendered to the Medical Association, the Natal Coastal Branch, and the public. The deep appreciation and thanks which he had earned were reflected in his election to the highest honour in the gift of the Association.

In reply, expressing his thanks and appreciation, Dr. Grant-Whyte said that in carrying out the duties of his office he would never wittingly stray from the path that had been blazed by men of good will, and that he would try to emulate his distinguished predecessors. He asked for the continued support of all members of the Association.

Dr. A. W. S. Sichel, in thanking Dr. du Toit for his distinguished services as President during the past year, said that he appreciated the opportunity of paying tribute to his old friend and colleague. They had been associated since their student days at Edinburgh, had entered the same speciality of ophthalmology, and on returning to Cape Town had worked together as close colleagues at the University Teaching Hospital, Cape Town. For many years they had served the Medical Association of South Africa (and its predecessor the British Medical Association), and now at the same time they were relinquishing the respective positions of President of the Association and Chairman of Federal Council, though they both hoped still to work in the interests of the Association. Dr. du Toit had served as Hon. Treasurer for over 30 years, and in his person had done much to hold the Association together by binding together sections that might have drifted apart. His record of service was a wonderful example and inspiration.

Dr. du Toit spoke briefly in reply.

At the opening of the meeting the minutes of the last annual general meeting were confirmed and signed and the annual report and audited accounts adopted. Messrs. Notcutt, Gurney and Fisher were reappointed as auditors.

The meeting was adjourned until 8 p.m. on 16 September, at the Town Hall, Durban, when Dr. Grant-Whyte delivered his presidential address at the opening ceremony of the South African Medical Congress (p. 945 of this issue).

FEDERAL COUNCIL MEETING

At the opening of the Federal Council meeting at the B.E.S.L. Hall, Old Fort Road, Durban, on 11 September 1957. Dr. J. H. Struthers, of Pretoria, was elected as Chairman of the Federal Council and inducted by Dr. A. W. S. Sichel, the retiring chairman, who had held the office for the past 12 years. Dr. Struthers' nomination was proposed by Dr. Sichel and seconded by Dr. T. Schneider, of Johannesburg. Dr. R. Schaffer, of Queenstown, presided during the election and induction.

Dr. Struthers, in thanking the meeting for the honour conferred on him and the confidence imposed, referred to Dr. Sichel's long term of office as a marvellous example of duty well done—which he would try to emulate. He was sorry that Dr. Sichel had decided not to stand again for election but was glad to know he was remaining on the Council and was willing to continue in active work.

The President of the Association (Dr. H. Grant-Whyte) paid tribute to Dr. Sichel for the distinguished manner in which, for so many years, he had served the Association in the position of Chairman of Federal Council. In Natal they held Dr. Sichel in extremely high regard; they had consulted him over their troubles and had always received excellent advice. Dr. Grant-Whyte said he had recently been present at the Representative Meeting of the British Medical Association at Newcastle-upon-Tyne and every one of the office-bearers had enquired about Dr. Sichel and expressed regret that he had not been able to be present.

Dr. J. S. du Toit said he had been associated with Dr. Sichel for half a century or more; they had both joined Federal Council in the early 30's. Dr. Sichel had had conferred on him the unique honour of the Presidency of the British Medical Association and was now Vice-president of the B.M.A. He had made a study of the constitution of the Medical Association of South Africa and was our greatest authority on that subject. Only those who had worked with him in Cape Town knew the enormous amount of time he had devoted to the affairs of the Association.

Dr. Sichel expressed his thanks to those who had spoken and to all who had supported him during his period of chairmanship. He recalled that he had been a member of Federal Council for 22 years and had never missed a meeting during that time. There were many reasons why he had felt it necessary to decline nomination for a further term of office. He said that the strain of office lay not so much in attending meetings as in dealing with problems that crop up between meetings—the responsibility was very great. Although he was no longer chairman of Federal Council he would remain *ex officio* member of Federal Council, the Executive Committee, the Head Office and Journal Committee and the Benevolent Fund Committee.

Dr. Sichel recalled that his predecessor Dr. J. H. Harvey Pirie had remained *ex officio* member of Federal Council throughout his term of office, though Dr. Harvey Pirie would have preferred to be relieved of the burden—his loyalty and diligence had been beyond all praise. A vote of thanks was passed to Dr. Harvey Pirie.

Vice-chairman. Dr. E. W. Turton, of Boksburg, was elected as Vice-chairman of Federal Council.

Vice-president. Dr. R. Schaffer, of Queenstown, was elected as Vice-president and President-elect of the Medical Association

of South Africa. Dr. Schaffer in response said that he regarded his election as a great honour to himself and the Border Branch. He would do his very best to follow in the footsteps of his illustrious predecessors.

Executive Committee. The following members were elected to the Executive Committee of Federal Council: Drs. B. A. Armitage (Natal Inland Branch), A. Broomberg (Natal Coastal), J. A. Currie (Cape Western), L. Robertson (S. Transvaal), R. Schaffer (Border), A. G. Sweetapple (Natal Coastal), R. Theron (O.F.S.), F. Ziady (N. Transvaal).

Federal Ethical Committee. The following members were elected to this committee: Drs. A. Broomberg, L. E. Lane, T. Schneider, A. W. S. Sichel, G. F. C. Troskie.

Central Committee for Contract Practice. The following members were elected: From S. Transvaal Branch—Drs. W. Girdwood, L. S. Robertson and J. Gluckman; Cape Western—T. B. McMurray, A. G. Paterson, J. H. L. Shapiro and A. A. Zabow; Natal Coastal—A. Broomberg and N. A. Rossiter; Northern Transvaal—J. H. Casewell and W. H. Lawrance; Border; L. Alexander; Cape Midland—A. P. Albert; East Rand—D. E. McKenzie; O.F.S.—D. Serfontein; Vaal River—W. Chapman; Other Branches—N. Kretzman.

Parliamentary Committee. The following members were elected: Drs. J. H. Casewell, C. M. Grundlingh, W. H. Lawrance, L. S. Robertson, J. H. Struthers, E. W. Turton, W. Waks, F. Ziady.

Honours. Four recommendations from Branches for the award of the Association's Bronze Medal were considered and approved, viz. to Dr. A. Broomberg of Durban, Dr. R. Schaffer of Queenstown, Dr. R. Theron of Bloemfontein and Dr. L. O. Vercueil of Florida. As there are only 2 remaining vacancies this year for the award of the Bronze Medal, lots were drawn, as the result of which Drs. Broomberg and Theron are receiving the medal during the present Medical Congress, and the presentation to Drs. Schaffer and Vercueil will stand over until next year.

On the recommendation of their Branches the following members were elected to Emeritus Membership of the Association: Dr. J. Drummond of Durban and Dr. C. Martiny of Johannesburg.

On the recommendation of the Head Office and Journal Committee the Hamilton-Maynard Memorial Medal for 1956 was awarded to Dr. Geoffrey Dean, of Port Elizabeth, for his paper entitled 'Porphyria, a Familial Disease: Its Diagnosis and Treatment', which appeared in the *South African Medical Journal* of 21 April 1956 on page 377. This medal is awarded to the author of the article in the *Journal* which is considered to be of the highest merit.

The Committee's recommendation was also adopted, that the Leipoldt Memorial Medal be awarded to Dr. H. Braudé, of Kroonstad, for his paper entitled 'Phenylketonuria' which appeared in the *Journal* of 28 January 1956 on page 83. This medal is awarded to the general practitioner whose article is considered to be of the highest merit.

DOCTORS WHO CANNOT BE TRACED

The Registrar of the South African Medical and Dental Council writes: Communications which had been addressed to certain medical practitioners by this Council were returned by the Post Office, as delivery could not be effected. Attempts to trace these persons have failed, and I shall therefore be glad if you will publish their names in your *Journal* with the request that they communicate with the Council without delay.

The attention of the medical practitioners whose names appear in the list subjoined hereunder, is invited to the following sections of the Medical, Dental and Pharmacy Act:

16 (2) 'It shall be the duty of every registered person who changes his address to intimate the fact to the registrar within one month after such change.

17 (1) 'The Council may erase from the register the name of any person who—

(*a*) .....

(*b*) has failed, within a period of three months from the date of an enquiry sent by the registrar by registered letter to the address appearing on the register in respect of him, to notify to the registrar his present address.'

Name	*Present Registered Address*
A. H. Antonis ..	204 Vredehoek, Grafton Road, Yeoville, Johannesburg.
S. M. Baker ..	X-ray Department, King Edward VIII Hospital, Durban.
B. O. L. Bettison ..	C/o Government Hospital, Greytown, Natal.
P. D. de Villiers ..	p/a Koning Eduard VIII Hospitaal, Durban.
C. P. Dickens ..	c/o Dr. F. V. Trotter, Nelspruit, Transvaal.
J. K. G. Grieves ..	'Sunninghill', Swartkopskloof, Pietermaritzburg.
J. B. Gurland ..	25 le Rhone Mansions, Regent Road, Sea Point, C.P.
M. R. D. Hart ..	71 Tregoning Street, Linksfield, Johannesburg.
L. Human	c/o South Africa House, Trafalgar Square, London, W.C. 2.
A. Kessler	304 Magaliesberg, cor. Louis Botha and Fife Avenues, Berea, Johannesburg.
J. K. Langenegger..	Krugersdorp Hospital, Krugersdorp.
J. A. McGregor ..	12 Seventh Avenue, Fish Hoek, C.P.
D. S. R. Morrison..	2 Tyson Road, Parkview, Johannesburg.
H. A. Nel	Angomahof 101, Vierdelaan, Germiston.
B. H. Osler.. ..	701 Burton Court, cor. Klein and Pretoria Streets, Hillbrow, Johannesburg.
B. Winokur ..	204 Vredehoek, Grafton Road, Yeoville, Johannesburg.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Tempogen

Tempogen represents the latest formulation of adrenocortical steroids in combination with salicylate, ascorbic acid and antacid. Messrs. Merck Sharp and Dohme supply the following information:

Tempogen provides lower steroid dosage with a higher level of symptomatic relief and extends the full benefits of steroid therapy to a wider range of patients—including many in whom he use of steroids alone may not be warranted.

Tempogen contains: Pred*nisolone* 1·0 mg. Acetylsalicylic acid 300 mg. Sodium ascorbate 60 mg. Aluminium hydroxide gel 200 mg. Prednisolone provides direct anti-inflammatory action. In combination with acetylsalicylic acid an additive steroid-salicylate action is provided. This combination avoids exceedingly large doses of both steroid and salicylate thus reducing side effects but in no way reducing the effective control of pain, stiffness and inflammation.

More sodium ascorbate is provided to give more positive protection against reduced adrenocortical and medullary tissue levels of Vitamin C as a result of stress or salicylate therapy. Hyperacidity and gastric distress often occur following the use of steroids or salicylates. The aluminium hydroxide gel in the formula protects against hyperacidity and is included in proper proportion according to the amount of steroid in the formula.

Dosage depends on the nature and severity of the condition being treated and the response to therapy. Dosage should be carefully individualized to provide control at the lowest dosage levels. Initial dose is one to four tablets 3 to 4 times daily. After satisfactory response gradually reduce to the lowest satisfactory maintenance dose.

Presentation: Tempogen Tablets in bottles of 10's and 30's.

Manufacturer: Merck Sharp & Dohne International Division of Merck & Co., Inc., P.O. Box 5933, Johannesburg.

Codelcortone-T.B.A.

Prednisolone tertiary-butylacetate. This product represents a radical extension and advance in *local* steroid therapy. Messrs. Merck Sharpe and Dohne supply the following:

Previously available steroid formulations for localized effect were confined to intrasynovial use. Codelcortone-T.B.A. is designed for *soft tissue injection* as well as intrasynovial injection. When making intrasynovial injection should some of the material accidentally be introduced into surrounding soft tissue no irritation occurs and indeed to the contrary beneficial local effect on the surrounding soft tissue results. Injections are also made directly into soft tissue.

Codelcortone-T.B.A. provides a wider range of local treatment with freedom from systemic effects extending over more prolonged periods of time and achieved at a lower dosage level. It is indicated in the treatment of tendinitis, trigger finger, Quervain's disease, tenosynovitis, Dupuytren's contracture, peritendinitis, ganglia, tennis elbow, lumbosacral strain, capsulitis, frozen shoulder, rheumatoid nodules, coccydynia, fibrositis, collateral ligament, strains and sprains, radiculitis and osteochondritis.

It is indicated for local control of pain, swelling, stiffness and inflammation in rheumatoid arthritis, osteoarthritis, chronic traumatic arthritides, acute gouty arthritis and in many types of bursitis.

Dosage: Detailed literature available on request.

Note: Intra-articular injection should not be done in the presence of specific infection. Intervertebral joints should *not* be injected. The only untoward effect is a transient post-injection flare which rapidly passes and occurs in one per cent or less of the patients and seldom recurs in the same patient.

Presentation: Codelcortone-T.B.A. 20 mg. per c.c. in 5 c.c. vials.

Manufacturer: Merck Sharp & Dohne International Division of Merck & Co., Inc., P.O. Box 5933, Johannesburg.

IN MEMORIAM

ELLY ISSEROW, L.R.C.P. & S. (EDIN.), L.R.F.P.S. (GLAS.) MB., CH.B. (EDIN.)

Dr. H. Penn of Johannesburg writes: Dr. Elly Isserow died in Johannesburg on 20 August 1957. He was 60 years of age. He graduated at Edinburgh University in 1921 and was in general practice in Marquard, O.F.S., from 1922 to 1935 and then in Johannesburg from 1935 until his death.

Dr. Isserow

His main interest was his profession which he loved intensely. He was a keen and active member of the Medical Association and rarely missed the clinical meetings. His desire for medical knowledge and the recent advances in medical practice was insatiable. His keen sense of duty and honesty of purpose was apparent to all. He took an especial interest in the care of the aged and his delightful sense of humour and remarkable personality were seen at their best under these circumstances.

He never spared himself and threw himself heart and soul into the personal conduct of his practice in spite of failing health during the latter years. It was a privilege to have known such a human and lovable personality. His death is a severe loss to the community and to the profession. Our deepfelt sympathy is extended to his bereaved wife and family.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. L. J. A. Loewenthal has been made a Corresponding Member of the Israeli Dermatological Association and an Hon. Member of the Venezuelan Dermatological Association.

* * *

Dr. J. G. Burger, Deputy Director of the Cape Provincial Hospital Services, has been appointed as the Medical Superintendent of Groote Schuur Hospital, to succeed Dr. N. H. G. Cloete, who is to retire at the end of October. Dr. Burger, who is 46, became Deputy Director of Hospital Services in February 1956.

* * *

The South African Society of Anaesthetists, Cape Western Branch. The next meeting of the Branch will be held on Tuesday, 24 September at 8.15 p.m. in the Small A-floor Lecture Theatre, Groote Schuur Hospital. Dr. H. Churchill-Davidson of St. Thomas' Hospital, London, will speak on *Muscle Relaxants and some Abnormal Responses to their use.* All interested are welcome.

* * *

Professor Norman M. Dott, C.B.E., F.R.S.E., F.R.C.S.E., Professor of Neurological Surgery, University of Edinburgh, will give an address on 'Subarachnoid Haemorrhage' at the Physiology Lecture Theatre, Medical School, University of Cape Town, on 27 September at 8.15 p.m. All medical practitioners are welcome. Professor Dott will give an address on 'Facial Pain' at the Harveian Lecture Theatre, Medical School, Johannesburg, on Thursday, 3 October 1957at 5.30 p.m. The lecture is open to all medical practitioners and students.

* * *

Union of South Africa. Department of Health. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 6 September-12 September 1957.

Poliomyelitis

	Eur.	*Nat.*	*Col.*	*As.*	*Total*
Transvaal	—	—	—	—	—
Cape Province ..	—	—	3	—	3
Orange Free State ..	—	—	—	—	—
Natal	—	—	—	—	—
	—	—	—	—	—
Totals	—	—	3	—	3

Plague. Two (2) fatal Native cases Bubonic plague in the Kibikwe reserve, portion of Morokweng district Vryburg.

Smallpox, Typhus Fever. Nil.

REVIEWS OF BOOKS : BOEKRESENSIES

CLEFT LIP AND PALATE

Cleft Lip and Palate. Second Edition. By W. G. Holdsworth, F.R.C.S. (Eng.). With a foreword by Sir Harold Gillies, C.B.E., F.R.C.S. Pp. xi + 187. 146 Figures. 42s. net. London: William Heinemann—Medical Books Ltd. 1957.

Contents: Preface. Foreword. Acknowledgements. I. The Lip and Palate. II. Development. By R. J. Harrison, M.A., D.Sc., M.D., B.Ch. III. Clefts. IV. Cleft Lip. V. Cleft Palate. VI. Secondary Operations. VII. Nursing Care. VIII. General Anaesthesia. By G. E. Ennis, F.F.A.R.C.S. and D. A. Sherman, M.D., D.A. IX. Orthodontic Treatment. By M. A. Kettle, F.D.S., D.Orth. R.C.S. Appendix—Suturing. References. Index.

This second edition improves on the high standard set by the original work. It is a very comprehensive review of everything concerning cleft lip and palate surgery, contains good diagrams and good operative descriptions, but it still has some minor shortcomings.

Some of the techniques described should be mentioned only historically and insufficient space is given to others, such as Tennison's operation for cleft lip.

The author should develop strong views to make his book still more useful for the young surgeon who needs the firm guidance resulting from experience and judgment.

D.S.D.

PHYSIOLOGY IN SURGERY

Physiologic Principles of Surgery. Edited by Leo M. Zimmerman, M.D. and Rachmiel Levine, M.D. Pp. xv+988. Illustrations. $15·00. Philadelphia and London: W.B. Saunders Company. 1957.

Contents: 1. Metabolic Changes Associated with Injury. 2. Infections and Antibiotics. 3. Thermal and Radiation Injuries. 4. Transplantation of Tissue. 5. Hemorrhage and Shock. 6. Blood Transfusion. 7. Body Fluids and Electrolytes. 8. Nutrition in Surgery. 9. Significance of Plasma Proteins in Surgical Practice. 10. Surgery of Newborn. 11. Pain. 12. Anesthesia. 13. Congenital Heart Disease. 14. Acquired Cardiac Lesions. 15. The Basis for Surgical Treatment of Hypertension. 16. Peripheral Arterial Vascular Disorders. 17. Venous and Lymphatic Systems. 18. Respiratory Tract: Physiology of the Thorax. Application of Pulmonary Function Studies to Thoracic Surgery. 19. The Esophagus. 20. The Stomach. 21. The Large and Small Intestine. 22. The Liver. 23. The Bile Tract. 24. The Pancreas. 25. The Blood and Blood-forming Organs. 26. The Thyroid Gland. 27. The Parathyroid Glands. 28. The Adrenal Glands. 29. The Islands of Langerhans: Hyperinsulinism. 30. The Kidneys. 31. The Lower Urinary Tract. 32. The Male Genital Tract. 33. The Uterus and Ovaries. 34. The Breast. 35. Kinetic Aspect of the Physiology of the Locomotor System. 36. Central Nervous System. Index.

Here is a book which cannot be reviewed adequately in the space usually available and all one can say is that it should be read by every doctor—and preferably by all medical students. Although edited by two eminent men, it is written by 50 contributors, each an authority in his subject. Consequently, each chapter is an entity in itself, complete with an extensive list of relevant references. The contributors have managed to apply their information to clinical and operative surgery, making the book an absolute delight to read.

There is, however, no easy bedside reading here, because even though the subject matter is cleverly handled, it is too extensive and comprehensive to be simple. Nevertheless, reviewing this book was a pleasure and it is a most welcome addition to my bookshelf, where it will not remain undisturbed for long.

D.J.duP.

WOUND SHOCK

Privy Council. Medical Research Council Memorandum No. 34. *The Treatment of Wound Shock—The Wound Shock Working Party.* Pp. vii+39. 4 Figures. 3s. 6d. Net. London: Her Majesty's Stationery Office. 1957.

Contents: Part I. Nature and Assessment. Introduction. Assessment of Patient's Condition. Conclusions. *Part II. Treatment.* General Measures. Restoration of Blood Volume by Transfusion. Operation. Post-operative Care. Metabolic Consequences of Injury. Special Complications of Injury. Burns. Toxic Gases. *Appendix I.* Transfusion Fluids provided by the Blood Transfusion Services in England, Wales, Scotland and Northern Ireland. *Appendix II.* Plasma Substitutes. *Appendix III.* Transfusion Apparatus. *Appendix IV.* Practical Points in the Intravenous Administration of Blood or Blood Products, including Grouping Tests. *Appendix V.* Untoward Reactions following Transfusion. *References.*

This excellent memorandum should be in the possession of every medical practitioner who is called upon to deal with wound shock. The working party has completely revised War Memorandum No. 1 on 'The Treatment of Wound Shock' and the present memorandum (No. 34) is absolutely up to date. Clear criteria for the assessment of the patient's condition are given and the treatment is described in detail. Special complications such as fat embolism and anuria are reviewed and the modifications for abdominal and chest injuries and for burns are outlined.

Appendices on transfusion fluids, plasma substitutes, transfusion apparatus, and reactions and practical points in the technique of transfusion, are a most valuable contribution to the memorandum.

The memorandum is recommended with the greatest enthusiasm.

J.H.L.

YEAR BOOK OF EYES AND E.N.T.

The Year Book of the Eye, Ear, Nose and Throat. (1956-57 *Year Book Series*). *The Eye.* Edited by Derrick Vail, B.A., M.D., D.Oph. (Oxon.), F.A.C.S., F.R.C.S. (Hon.). *The Ear, Nose and Throat.* Edited by John R. Lindsay, M.D. Pp. 448. 128 Illustrations. $7·00. post paid. Chicago: Year Book Publishers, Inc. 1957.

Contents: Publisher's Note. *The Eye.* The Orbit and Adnexa. The Conjunctiva and Cornea. The Uvea. Retraction and Motility. The Lens and Cataract. Neurology and Visual Fields. The Retina. Glaucoma. Surgery. Therapy. Miscellaneous. *The Ear.* Vestibular Function and Vertigo. Hearing and Hearing Tests. Tubal Function and Inflammatory Ear Disease. Otosclerosis. Fenestration and Stapes Mobilization. Miscellaneous. *The Nose and Throat.* The Nose and Sinuses. The Mouth, Oropharynx and Nasopharynx. Salivary Glands. The Larynx and Neck. Trachea, Bronchi and Tracheotomy. The Hypopharynx and Esophagus. Allergy. Acute Respiratory Disease (ARD) Viruses. Miscellaneous.

In a book of this type the reviewers are at a disadvantage, for they have to read in a hurry and consequently have not the leisurely approach permitting absorbtion and retention of so much unconnected material. This is no book to be consumed in one sitting, but rather a bedside delicacy to be nibbled at odd moments, a few pages at a time. Like its predecessors it consists of summaries of the important articles that have appeared in the world literature for the period between October 1955 and September 1956. This is to be the last edition combining the Eye and the ENT sections. From next year they will be independent, thus permitting an extension of each subject. It is hoped that the quality of the paper, print and illustrations will also be maintained.

The summaries are considerably longer than an abstract, such as those of the American Journal of Ophthalology, and more detail is therefore possible. Also most of the articles have an appended critical review by the Editor. The criticisms may vary from a 'wow' (chorio-retinal burns are obtainable 42·5 miles from an exploded atom bomb) to several sentences, but in all cases are constructive. It is felt that the book serves the purpose of enabling one to keep the previous year's work fresh in mind, there being a tendency for this to become neglected in the rush to keep right abreast with the spate of the more recent journals.

L.S.

This year's edition maintains its interest as an excellent resumé of the world literature with succinct editorial comment after each article. For practitioners with an interest in this field it is an economic method of covering the year's trends. It is as usual, an excellent, clear and well-illustrated publication.

The section on vestibular function and vertigo gives a good review of modern concepts; of special interest is postural and positional vertigo. Psychogalvanic skin reactions in audiometry are regarded as of doubtful value. The present status of fenestration and stapes mobilization in otosclerosis achieves a prominent place. Tympanoplasty is of marked interest and there are excellent articles by Wullstein and Zöllner.

The sections on the nose are mainly interested in the plastic field.

The changes in the other sections are not marked, but the use of continuous negative-pressure wound drainage after laryngectomy (post-irradiation) is of interest.

In the allergy section, it is suggested that food sensitivity is important in perennial vasomotor rhinitis in adults. Steroids in nasal allergy are condemned except for local use.The statement that over 90% of chronic sinusitis is basically an allergic rhino-

Sinusitis with or without secondary infection is acceptable in this country.

Articles on the adenoid-pharyngeal-conjunctival viruses and Cocksackie Group-A viruses are of special interest.

B.G.

CANCER

Cancer. Volume I. Part I. Research into Causation. Edited by Ronald W. Raven, O.B.E. (Mil.), T.D., F.R.C.S. Pp. x+539+(19). 94 Figures. 85s. + 2s. 2d. delivery. (Complete work will consist of 6 Volumes and Index). London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

Contents: Preface to the Work. Contributors to this Volume. *Part I: Research into Causation.* 1. Historical Aspects of Cancer. *W. R. Bett.* 2. The Incubation Period of Cancer in Man. *E. L. Kennaway.* 3. Carcinogenic Effect of Cholesterol. *E. L. Kennaway.* 4. Carcinogenesis. *P. R. Peacock.* 5. Chemical Mechanisms of Normal and Abnormal Cell Division. *J. N. Davidson.* 6. Biochemistry of Cancer Induction. *A. Clark Griffin.* 7. Hormones and Neoplasia. *Michael B. Shimkin.* 8. The Part Played by Viruses in the Origin of Tumours. *Leon Dmochowski.* 9. Nutrition and the Genesis of Tumours. *Albert Tannenbaum and the late Herbert Silverstone.* 10. The Genetic Component of Cancer. *P. C. Koller.* 11. Environmental Factors in the Production of Human Cancer. *W. C. Hueper.* 12. Carcinogenic Effects of Radiation. *A. Glucksmann, L. F. Lamerton and W. V. Mayneord.* Index.

This volume is a work of outstanding merit not only as a book which fulfils many criteria (size, weight, binding etc.) but also in the manner in which the subject matter is presented. The text is liberally supported by charts, tables, diagrams and reproductions that are equally commendable. One criticism in this regard may be levelled at the glossiness and whiteness of the paper, which makes reading a little uncomfortable.

The chapters flow naturally one into the other in a manner that flattens out the effects of the discontinuities so prone to arise in publications of this type. The reader not unexpectedly senses the weight of authority behind each statement, for the contributors have been specifically selected from those most renowned in the English-speaking world. Such a standard is maintained throughout that it would be quite inappropriate to single out any chapter for individual comment or praise. The significant feature is the breadth of vision and the intrinsic balance of the chapters and of the work as a whole.

Obviously, in so comprehensive a work, where our knowledge is in a state of flux, certain lines of approach may appear initially to be under- or over-emphasized. This, however, is only the first in a series of volumes and supplements where, no doubt, such apparent discrepancies will later receive due attention.

The vast amount of research that has been and is being undertaken is revealed not only in the body of each chapter but also in the very comprehensive references (up to 600 in some cases) at the end of each chapter.

Perhaps a fitting description of the work would be: a reference book one is loath to put down. It should therefore be found in every medical library and in the personal library of everyone interested in cancer.

J.M.G.

BIOCHEMISTRY

Kinetics and Thermodynamics in Biochemistry. By H. Geoffrey Bray, D.Sc. (Birm.) and Kenneth White, B.Sc. (Lond.), Ph.D. (Birm.), F.R.I.C. Pp. xii+343. 71 Illustrations. 42s. net. London: J. & A. Churchill Ltd. 1957.

Contents: Preface. Symbols, Abbreviations and Conventions. 1. Atoms and Molecules. 2. Particles in Motion. 3. Principles of Thermodynamics. 4. Equilibria in Aqueous Solution. 5. The Rate of Reaction. 6. The Kinetics of Reaction Systems. 7. The Kinetics of Enzymic Reactions. 8. Free Energy and Metabolism. 9. Kinetics and the Whole Organism. Index.

This book represents a systematic development of the basic ideas of kinetics and thermodynamics as applied to biochemistry. It is written primarily for the honours and post-graduate student and the treatment of the subject matter is thus necessarily at a somewhat advanced level. The mathematical knowledge required, however, does not extend beyond an acquaintance with the ideas of simple calculus.

The topics discussed include structure and biochemical function, energetic coupling and active transport, equilibria concerning oxidation-reduction reactions and phosphate compounds, the kinetics of reaction systems, isotopic tracers in reaction systems, enzyme kinetics, pathways of free energy, and control of metabolism.

The book will appeal to biochemists and physical chemists alike and will do much to foster the cross-fertilization between those sciences which seek to elucidate the behaviour of living matter.

The book fills an important gap in biochemical literature by collecting much useful material which would otherwise be scattered amongst specialist text-books and journals. It is an invaluable contribution to our knowledge of the major growing points of modern biochemistry.

H.Z.

DRUG THERAPY YEAR BOOK

Year Book of Drug Therapy—1956-57 *Year Book Series.* Edited by Harry Beckman, M.D. Pp. 514+70 Figures. 86·75. Chicago: Year Book Publishers, Inc. 1957.

Contents: Publisher's Note: Introduction. Allergic Disorders. Cardiovascular Diseases: Arrhythmias, Congestive Heart Failure, Hematologic Coronary Disease, Hypertension, Peripheral Vascular Diseases, Thrombosis, Thrombophlebitis and the Anticoagulant Drugs. Chest Diseases. Corticotropin and the Cortisones. Dermatologic Maladies. Diabetes Mellitus. Gastroenterologic Disorders. Growth Disturbances. Hematologic Disorders. Hypothyroidism. Infectious Diseases: Antibiotics and Sulfonamides—General Considerations, Miscellaneous Infections, Urinary Tract Infections. Tuberculosis. Kidney Disorders. Lead and Organic Phosphate Poisoning. Liver Disorders. Neoplastic Diseases. Neuropsychiatric Disorders. Obesity. Obstetric and Gynecologic Disorders. Ophthalmologic Disorders. Pain. Rheumatic Disorders. Surgical Disorders. Thyrotoxicosis. Urolithiasis. Worm and Fluke Infestations.

The Year Book series is well known to all doctors. The high standard and valuable features are maintained from year to year. There is the usual large number of abstracts of articles on a variety of topics (see table of contents above); it is the American medical literature that is principally represented. Many of the articles are from journals not easily available to all in this country. There are numbers of charts and tables, and an author index as well as the general subject index. Critical editorial notes and comments are a valuable feature of the service provided in these books.

Drugs of many types that have undergone clinical trial over the last year or two are presented for further study by the reader. Those practitioners with research interest will see what has been happening and may be stimulated to conduct studies of their own.

A large number of articles deal with the subject of drugs which affect mood and behaviour (tranquillizing agents), half of the articles being chosen deliberately by the editor to emphasise the toxic proclivities of these extensively prescribed agents. The section on anticoagulants is preceded by authoritative answers to 15 important questions relating to their use and should be studied by everyone using these drugs. The newer anticholinergic drugs proposed as substitutes for atropine in the treatment of peptic ulcer have not displaced this old drug. Ready-mixed proprietary preparations, as marketed for instance in the case of anti-hypertensive drugs, are deplored because the physician loses the advantage of adjusting the dosage of the individual ingredients. Attention is drawn to the ever-changing remedies for trichomonas infection. A warning is given regarding the use of folic acid with cyanocobalamin in pernicious anaemia, which may be harmful. The mode of action of aspirin (incidentally holding its own against cortisone in rhematoid arthritis) is still unexplained. These examples should serve to illustrate some of the many investigations and unsolved problems of therapy reported by clinical workers and presented in condensed form for the interested reader.

N.S.

TREATMENT OF HYPERTENSION

Die Behandlung Des Bluthochdruckes. Von Prof. Dr. L. Hantschmann. viii+92 Seiten. Abbildungen. DM 12.-. Stuttgart: Georg Thieme Verlag. 1956.

Inhaltverzeichnis: A. *Diagnose und Differentialdiagnose des Bluthochdrucks.* I. Diagnose der Blutdrucksteigerung. 1. Die Blutdruckmessung. 2. Werte und Grenzen des normalen Blutdrucks. 3. Feststellung der krankhaften Blutdruckerhöhung. II. Differentialdiagnose der Blutdrucksteigerung. 1. Die Bedeutung der Differentialdiagnose für die Therapie. 2. Die Einteilung nach kreislaufmechanischen Gesichtspunkten. 3. Die Einteilung nach klinischen Gesichtspunkten. 4. Die Blutdrucksteigerung bei Erkrankungen des Herzens und der grossen Blutgefässe. 5. Der zentral-nervöse Hochdruck. 6. Der endokrine Hochdruck. 7. Hochdruk bei Infektionskrankheiten. 8. Der renale Hochdruck. 9. Der Schwangerschaftshochdruck. 10. Die essentielle Hypertonie. III. Diagnose

des Schweregrades der Hypertonie. B. *Therapie*. I. Die Hypertonie bei Erkrankungen des Herzens und der grossen Blutgefässe. II. Der zentral-nervöse Hochdruck. III. Der endokrine Hochdruck. IV. Hochdruck bei Infektionskrankheiten. V. Der renale Hochdruck. VI. Der Schwangerschaftshochdruck. VII. Die essentielle Hypertonie. (*a*) Ziele und Möglichkeiten der Behandlung. (*b*) Beeinflussung der psychischen und nervösen Faktoren. (*c*) Körperliche Tätigkeit, Sport und physikalische Massnahmen. (*d*) Diät. (*e*) Medikamentöse Behandlung. (*f*) Röntgentherapie. (*g*) Die chirurgische Behandlung. (*h*) Zusammenfassende Leitsätze zur Hypertoniebehandlung. VIII. Die Behandlung der Folgezustände des Bluthochdrucks. (*a*) Herz. (*b*) Blutgefässe. (*c*) Gehirn. (*d*) Nieren. Schrifttum. Sachregister.

This is an excellent and comprehensive book on the aetiology and treatment of hypertension. The subject is extremely well presented and, although the major portion of the book is devoted to the treatment of essential hypertension, the importance of other causes of hypertension is emphasized so that appropriate measures can be instituted for the removal of the specific aetiological factor.

All the usual causes of hypertension are listed, i.e. coarctation of the aorta, unilateral and bilateral renal disease, arteriovenous fistulae, phaeochromocytoma and toxaemia of pregnancy, as well as endocrine causes, central nervous diseases and the hypertension associated with infectious diseases. The appropriate treatment in each case is fully discussed.

This is an essentially practical book, full of basic valuable information, written by a man with wide clinical experience. We are reminded that measures to reduce the blood pressure are not indicated in every case of hypertension and may in fact be harmful in those instances where the elevated blood pressure is due to an increase in stroke volume of the heart, as in heart block, aortic incompetence, arteriovenous fistulae and marked arteriosclerosis of the great vessels.

A full and critical account is given of the various therapeutic measures that have been employed in the treatment of hypertension. Among the ones that have stood the test of time, weight reduction, salt-free diet and rearrangement of the patient's life to allow for more leisure and less nervous tension, are still of prime importance and are by no means superseded by modern hypotensive drug therapy. In the discussion of the various therapeutic agents, stress is laid not only on their hypotensive properties, but also on the side-effects and hazards of administration of the more potent drugs, a point of special importance in out-patient treatment.

The various sympathectomies, adrenalectomy and the treatment of the cardiac, cerebral and renal complications of hypertension are briefly discussed.

This is a concise and useful book which can be read with advantage both by students and doctors alike. It is written in German.

H.L.

THE ELDERLY SICK

The Care of the Elderly Sick in General Practice. Being the John Matheson Shaw Lecture for the year 1956 delivered in the Hall of the Royal College of Physicians of Edinburgh on November 9th, by W. Ferguson Anderson, M.D., F.R.F.P.S. Glasg., M.R.C.P. Lond. Adviser to the Western Regional Hospital Board (Scotland) in Diseases of Old Age and Chronic Sickness; Honorary Lecturer in Materia Medica in the University of Glasgow. Pp. 27. Edinburgh: The Royal College of Physicians. 1957.

This booklet—a primer in Geriatrics—comprises the 1956 John Matheson Shaw Lecture of the Royal College of Physicians of Edinburgh delivered by Dr. W. Ferguson Anderson. It is brimful with Experience—tested, reliable information on varied subjects ranging from the pitfalls in diagnosis of coronary thrombosis and appendicitis to the bugbear of geriatrics—bedsores. Helpful advice is given on mental aberration, gastro-intestinal disturbances, osteoporosis, hemiplegia, insomnia, surgery, and a host of other subjects. An apt plea is made for the reinstatement of careful history-taking as a valuable aid in diagnosis—a timely antidote to laboratory fanaticism.

And all this is dealt with in barely 23 easily read pages; a remarkable achievement made possible by avoiding padding, platitudes and similar space-consuming extravagances.

This lecture should serve to remind one that many easily applicable means for relieving suffering and countering disability in the aged are readily available, and should whet the reader's appetite for further study of the subject of geriatrics.

I.M.H.

STUDENT'S HISTOLOGY

A Student's Histology. By H. S. D. Garven, B.Sc., M.D. (Glas.), F.R.S.E., F.R.F.P.S.G. Pp. xii + 650. Illustrations (Some in colour). 55s. net. + 1s. 6d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

Contents: Preface. To the Student. List of Contractions. 1. The Cell and General Considerations. 2. The Tissues of the Body. 3. Glands and Secretory Activity. 4. The Circulatory System. 5. Haemocytopoietic and Lymphatic Organs. 6. The Respiratory System. 7. The Skin. 8. The Digestive System. 9. Excretion. 10. Integration—The Endocrine Organs. 11. Reproduction. 12. Integration—The Reflex Arc. 13. The Special Senses. *Appendix I.* Suggestions on Further Reading. *Appendix II.* A List of Histologists and Others. Index.

For many years there has been a marked gap in the medical student's armamentarium of text books on physiological histology. The available texts have either been very elementary or have dealt with the subject in sufficient scope and detail for the advanced student or research worker.

This new book by an outstanding teacher and histologist fills the gap in a remarkable manner. The clarity of the text, both visually and conceptually, and the organization of the book, leave little to be desired. The plates and figures, all of which are new, must be particularly commended.

An interesting innovation is notes on the Latin and Greek derivations of the names of the major structures and organs discussed. Eponymous nomenclature, although still used, is commendably reduced in this volume. The name of Malpighi, for example, is no longer perpetuated in the kidney or in the skin. In this respect the 'List of histologists and others' in Appendix II is of great interest and value.

The correlation of structure and function is, on the whole, well covered by Dr. Garven. He avoids the extremes of paucity or over-discussion of physiological associations seen in some of the more expensive volumes. The reasonable cost of this book, as text-books go, has been achieved without reduction in the quality of type face or finish, both of which are excellent.

Dr. Garven's book cannot be too highly recommended to all students of medicine and biological science. It will also be of considerable help to those studying for specialist qualifications.

A.G.R.

CLINICAL TOXICOLOGY

Clinical Toxicology. The Clinical Diagnosis and Treatment of Poisoning. By S. Locket, M.B., B.S., M.R.C.P. (London). With Special Sections on the Identification and Estimation of Some Common Toxic Substances by W. S. M. Grieve, M.Sc., Ph.D., F.R.I.C. and the Identification and Botanical Characteristics of Some of the More Frequently Encountered Poisonous Plants by S. G. Harrison, B.Sc. Pp. xii + 772. 27 Illustrations, including 2 coloured plates. £5 5s. net. London: Henry Kimpton. 1957.

Contents: Preface. I. Introduction. Facts and Figures. *Part I. Basic Treatment in Poisoning.* Principles and Practice Discussed in respect to Injury and Damage to Individual Organs and Systems. II. The Immediate Treatment of Acute Poisoning; Gastric Lavage; Warmth; Posture; Sedation; Nausea and Vomiting; Diarrhoea and Colic. III. Disorders of the Respiratory System (including Acute Respiratory Failure; Artificial Respiration; Pulmonary Oedema; Oxygen and Carbon Dioxide; and Acute Heart Failure). IV. Disorders of Fluid and Salt Balance; of the Kidney and Renal Tract occurring in Poisoning; Shock and Peripheral Circulatory Failure (including Anuria; Intravenous Fluids; Dehydration; Water Intoxication; Potassium Intoxication; Hypercalcaemia and Renal Failure). V. Liver Damage; Anticoagulants; Dextran. VI. Disorders of the Blood and Haemopoietic System. VII. Antibiotics; Sulphonamides, Sulphones and Diamidines; Hypersensitivity; Antidotes (including "B.A.L." "E.D.T.A." and Nalorphine Hydrochloride); Pesticides and Synthetic Detergents. *Part II. Symptoms and Treatment Described for Individual Poisonous Agents.* VIII. Toxic Gases. IX. Sedatives, Hypnotics and Narcotics, Section I (including Barbiturates; Sulphonal; Urethane; Primidone; Phenylacetylurea; Ureides and Bromides; Phenytoin; Troxidonum and Methylpentynol). X. Sedatives, Hypnotics and Narcotics, Section II (including Opium; Morphine and its derivatives; Pethidine; Amidone; Phenadoxone; Cannabis Indica and Mescal; Drug Addiction). XI. Alcohols, Ethers, Aldehydes, Ketones, Esters and Glycols, Section I (including Ethyl Alcohol; Acute Alcoholic Intoxication; Chronic Alcoholism; Antabuse and Dithiocarbamic Acid Derivatives). XII. Alcohols, Ethers, Aldehydes, Ketones, Esters and Glycols, Section II (including the Halogenated Aliphatic Hydrocarbons). XIII. Salicylates; Phenacetin and related Aromatic Analgesics; Phenylbutazone; Quinine; Quinidine and related Compounds; Mepacrine and other Anti-malarials. XIV. Convulsants (including Cocaine and the Local Anaesthetics and the Antihistaminics). XV. Vegetable and Plant Poisons, Section I. XVI. Vegetable and Plant Poisons, Section II. XVII. Vegetable and Plant Poisons, Section III. XVIII. Venomous Animals. XIX. Phenols, Cresols, and their Derivatives;

DNC and related Compounds; Naphthalene and its Derivatives; Aniline and the Aromatic Amines; Nitro-benzene. XX. Corrosive Acids, Alkalis and related Compounds. XXI. Arsenic; Antimony; Bismuth; Phosphorus; Lead. XXII. Mercury and its Compounds; Beryllium; Cadmium; Iron; Gold; Zinc; Manganese; Thallium; Nickel; Tellurium; Vanadium; Aluminium; Copper; Cobalt; Magnesium; Osmium; Platinum; Silver; Tin and Lithium. XXIII. Radioactive Substances; Atomic Bomb. XXIV. Toxic Effects of a Miscellaneous Group of Drugs and Therapeutic Agents including Hormones and Vitamins. *Part III. Special Sections on Identification of Poisons and Poisonous Plants.* XXV. The Identification and Estimation of some Common Toxic Substances. (W. S. M. Grieve, M.Sc., Ph.D., F.R.I.C.). XXVI. The Identification and Botanical Characteristics of some of the more frequently encountered Poisonous Plants. (S. G. Harrison, B.Sc.). Index of Treatment. Index of Authors. Index of Plant Names. Subject Index.

There are many smaller or larger books on the subject of toxicology. This is a new book with the emphasis on the fact that it is written by a physician in active clinical practice with a unit under his care that is primarily concerned with the treatment of poisoning. Their experience in the field of clinical toxicology they regard as almost unique. The subjects considered in this volume include therapeutic agents, chemicals used in industry and in warfare, venous and poisonous plants. Most of the information is derived from studies in human beings. Alcoholism and addiction and suicidal agents are considered in special detail. The book is divided into 3 parts. Part I deals with the basic treatment of poisoning, principles and general measures. In part II treatment is described for each individual toxic agent or group of agents. Part III contains two sections in which the identification of some commonly occurring toxic substances and poisonous plants and seeds are considered. There are numerous references to the literature, and a comprehensive index (treatment; authors; plants; subject index) containing more than 7,500 entries.

This book will be of value to medical and non-medical workers interested in the clinical diagnosis and treatment of poisoning.

N.S.

THERAPEUTICS

The Principles of Therapeutics. By J. Harold Burn, M.A., M.D., F.R.S. Pp. ix + 278. 36 Figures. 27s. 6d. Oxford: Blackwell Scientific Publications. 1957.

Contents: I. The Development of Therapeutics. II. Ion Exchange in Cellular Activity. Active Substances in the Body. III. Transmission of Nerve Impulses by Acetylcholine and Noradrenaline. IV. The Properties of Acetylcholine and of Substances modifying its Action. V. The Treatment of Hypertension. VI. Substances whose Action resembles that of Acetylcholine, especially Nicotine. VII. Adrenaline and the Sympathomimetic Amines. VIII. The Action of Histamine. Anaphylaxis, Allergy. Histamine Liberators and Anti-Histamine Substances. IX. Hydroxytryptamine and Bradykinin. X. Pituitary (posterior lobe) Extract. Biological Standardization. XI. The Digitalis Glycosides. XII. Substances relaxing Smooth Muscle including Vasodilators. XIII. Analgesic Substances. XIV. Cocaine and the Local Anaesthetics. XV. Alcohol. XVI. Anaesthetics. XVII. Barbiturates and Tranquillizing Drugs. XVIII. Stimulant Drugs. XIX. Drugs on the Alimentary Canal. XX. Action of Drugs on the Uterus. Ergot. XXI. The Treatment of Anaemias. XXII. The Thyroid Gland. XXIII. Insulin. XXIV. Oestrogens and Androgens. XXV. Adrenal Cortex. XXVI. Disinfectants. XXVII. Sulphonamides Salicylates. XXVIII. The Kidney. Treatment of Oedema. XXIX. Antibiotics. XXX. Malaria. XXXI. Chemotherapy in Tropical Diseases. XXXII. Metallic Salts and Vitamins. XXXIII. Biological Variation and Clinical Trials. Index.

This book contains the lectures on pharmacology and the basis of therapeutics given to medical students at Oxford. It is not a text-book of pharmacology nor yet of therapeutics; but a knowledge of its contents will enable the student to use his pharmacology when treating his patients and the physician to maintain his contact with the experimental observations on which his treatment is based. It is recommended to those who want brief notes on the principles involved in the therapeutic use of drugs.

Experimental evidence is given for the combined use of ephedrine with neostigmine in myasthenia gravis; for the hypotensive action of reserpine (dislodging 5-hydroxy-tryptamine in ganglia and thus blocking transmission); for the effect of cigarette-smoking on the liberation of antidiuretic hormone or vasopressin; for the production of essential hypertension by dopamine, through the failure of amine oxidase to destroy this pressor substance. Numerous other views on the mode of action of drugs, both established and tentative, are clearly presented and often illustrated by figures and tracings. It is surprising to read a description of the use of thallium acetate for ringworm without mention of its great toxicity. Dimercaprol is not of value in lead poisoning. There is no mention of the effect of orally administered anticoagulants on factor VII, nor is there indication of the chemo-receptive emetic trigger zone, nor of the action of iodine on thyrotrophic hormone and the thyroid proteolytic enzymes in hyperthyroidism. Diiodohydroxyquinoline might be mentioned in the treatment of amoebiasis. There are better alternatives to filix mas than pelletierine and cousso. On p. 257 'parathyroid' should be read for 'paratyphoid'.

This is an interesting and useful book for students and physicians.

N.S.

CORRESPONDENCE : BRIEWERUBRIEK

CHURCH SERVICE FOR DOCTORS

To the Editor: May I ask the hospitality of your columns to draw the attention of members of the medical profession to the special service for Doctors to be held in St. Stephen's Church, Pinelands, on Sunday, 20 October, at 7.30 p.m.?

A similar service has been held in the last two years and has been so warmly welcomed by so many that I feel another service this year is justified.

The purpose of such a service is twofold. On the one hand I hope it will provide an opportunity for many engaged in the practice of medicine to come together to unite in a service of prayer and dedication for the work in which they are engaged and the patients with whom they are concerned.

On the other hand I hope that many who are profoundly conscious of a deep debt of gratitude for the benefits they have received through the ministrations of doctors and nurses will find in this service an occasion when they too may gather together to give thanks and pray for those through whose skill and devotion they have received so many blessings.

Leslie A. Davis
Rector

The Rectory
Central Square
Pinelands
5 September 1957

XYLOCAINE INTOXICATION

To the Editor: While wishing to congratulate Drs. Bingham and Malherbe on their excellent report[1] of three cases of Xylocaine intoxication, I feel that a hasty perusal of their paper may lead your readers to believe that Xylocaine is an unsafe drug.

May I stress that in the dosage recommended by Moore[2] toxic reactions must be very rare indeed. In the large non-European hospital where I work Xylocaine has enjoyed a deserved popularity over the last two years and the only untoward reactions noted have been due to proved overdosage. I have never personally seen or heard of any fatal reaction following Xylocaine in the recommended dosage nor does the literature on the subject reveal any such case. I should be interested to hear of any toxic reactions to Xylocaine in a dosage of 3 mgms per lb. body weight or less.

May I remind your readers that no advantage accrues from using a greater concentration than 1% for nerve blocks or 0·5% for local infiltration. Moreover an 0·25% solution can be used for local infiltration with consistently good results particularly in the young, the old and the ill. Due to the fact that Xylocaine is not a vasodilator (cf. procaine) adequate vasoconstriction is obtained by an adrenaline concentration of 1 : 500,000.

The conclusion must be reached that Xylocaine is the safest and most effective local anaesthetic agent at present available to us.

A. J. Cilliers

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1. Bingham, L. R. and Malherbe, P. H. (1957): S. Afr. Med. J., **31**, 882.
2. Moore, D. C. (1955): *Complications of Regional Anaesthesia.* Oxford: Blackwell.